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PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

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J. Y. W. MACALISTER
UNDER THE DIRECTION OF
THE EDITORIAL COMMITTEE

VOLUME THE EIGHTH
SESSION 1914-15

PART III

ODONTOLOGICAL SECTION SECTION OF OPHTHALMOLOGY
OTOLOGICAL SECTION PATHOLOGICAL SECTION
SECTION OF PSYCHIATRY SURGICAL SECTION
THERAPEUTICAL AND PHARMACOLOGICAL SECTION

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1915

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PROCEEDINGS
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ROYAL SOCIETY OF MEDICINE

VOLUME THE EIGHTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1914-15

ODONTOLOGICAL SECTION



LONDON
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1915

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Odontological Section.

November 23, 1914.

Dr. A. W. W. BAKER, President of the Section, in the Chair.

The Influence of Nasal Obstruction on Abnormalities of the Jaws.

By H. LAMBERT LACK, M.D.

I FEEL I ought to apologise for bringing such a well-worn, almost threadbare, subject before you for discussion; but I have submitted my remarks to your Secretary, therefore you must blame him and not me. In spite of knowing what I am about to say, he persisted in inviting me here, and I was only too ready to accept such an honour. I fear I have little to say that is new, therefore I will be brief and give merely an outline of the views I hold.

The deformities of the upper jaw that can be ascribed to adenoids or other forms of nasal obstruction are:—

(1) Deficient development, which results in overcrowding and irregularity of the teeth.

(2) Lateral compression, which results in a V-shaped alveolar arch and in a high, steep, narrow palate.

These two conditions are generally associated and are accompanied by a similar but less marked deformity of the lower jaw. In the majority of cases there is an actual raising of the roof of the mouth, although this has been denied. The raising of the palate, and in consequence the raising of the floor of the nose, is shown by the deflection of the nasal septum. The degree of this deflection usually

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corresponds exactly with the raising of the palate. There are exceptions to this. There may be a high, narrow palate *without* a corresponding deflection of the septum, and then it will be found that the lateral compression of the jaw has resulted in a lowering of that portion of the alveolus containing the molar teeth. As a result of this, when the mouth is closed the molar teeth of the upper and lower jaws will come in contact earlier than normal and prevent the incisor teeth meeting—the condition of “open bite.” This condition of open bite is often associated with, and exaggerated by, deficient development of the front of the upper jaw in the vertical direction. In practice these conditions cannot always be sharply determined. Very often a patient may exhibit signs of all three varieties of deformity. Thus a high, narrow palate may be due partly to raising of the palate, as shown by septal deflection, and partly to lowering of the molars, as shown by the open bite, neither the open bite nor the septal deflection alone being as marked as one would expect from the height of the palate. Also, an open bite is often due to a combination of lowering of the molars *plus* want of growth of the pre-maxilla.

Repeated observations have convinced me that the characteristic deformity, the lateral compression of the upper jaw, is due to one cause alone—viz., nasal obstruction sufficient to cause mouth-breathing. The open mouth puts the soft tissues of the cheeks upon the stretch; this increased tension exercises a lateral compression upon the jaws. When the mouth is open there can be felt tense bands in the cheeks corresponding roughly to the naso-labial folds. It might be objected that the force exerted by this tension is extremely slight, but an almost imperceptible pressure constantly in action will readily mould a living structure. The rapid way in which one can remedy in a few days or weeks the result of years demonstrates this, for the open mouth takes years to produce its effect. The arguments in favour of this causation are briefly as follows:—

The point of greatest compression of the jaw exactly corresponds to the position of the above-mentioned bands; almost every specimen shows that the moulding is greatest about the position of the second bicuspid. The increased tension of the cheeks is the only force which would act upon both upper and lower jaws. No mouth can be kept open without increasing the tension of the cheeks, and no child can maintain an open mouth for years without the deformity occurring. The longer the mouth has been open the greater the deformity; one can form a fairly accurate opinion as to the time a child has kept its

mouth open by the amount of the compression of the jaw. It follows that patients with complete congenital nasal obstruction should show the greatest deformity, and this is the case, as notably seen in cases of bilateral congenital choanal atresia. A convincing proof of these theories was furnished by a patient attending my clinic. A boy, aged 12, suffering from nasal obstruction due to adenoids, had been a mouth-breather for many years. Since the age of 2 years he had been completely paralysed on one side of the face. The result of this and the open mouth was to render the cheek on the sound side tense, whilst the paralysed cheek remained flaccid. The effect on the jaw was obvious. On the flaccid (paralysed) side the jaw was well developed; the alveolar arch was normal. On the sound, tense side of the cheek the alveolar arch was flattened; the palate was raised. There could be only one possible explanation: the deformity was unilateral, because the compressing force on one side was absent owing to the flaccidity of the paralysed cheek. If the narrowing of the upper jaw was due to mouth-breathing simply, to ill-development of the nose owing to want of nasal respiration, or to any of the other causes often assumed, the deformity should have been bilateral. I showed this patient at the Laryngological Society of London¹ and drew attention to the overwhelming proof afforded by this single case, the one natural experiment which completely demonstrates the theory. I was very glad that some few years later an exactly similar case was discovered in Glasgow—unilateral facial paralysis with mouth-breathing which resulted in unilateral deformity of the jaw. No more convincing proof could be desired.

Another series of cases may be mentioned which disprove those theories which ascribe the deformities of the jaw to (1) the effect of the air passing through the mouth, (2) absence of air passing normally through the nose, (3) to diminished air tension in the nose, and so forth. In cases of *unilateral* congenital choanal obstruction the affected nostril is often as well developed as the unobstructed one. The deformity of the upper jaw varies. If the unaffected nostril is sufficiently clear to admit the patient to breathe through it without opening the mouth the upper jaw will be normally formed; if the clear nostril is not sufficient for the patient's respiration but mouth-breathing is also necessary, then there will be more or less lateral compression of the upper jaw; but in every case this has been found to be symmetrical and not unilateral.

¹ *Proc. Laryng. Soc. Lond.*, 1904-1905, xii, p. 95.

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The *deficient development* of the upper jaw is a more difficult condition to explain satisfactorily. That there is deficient development we probably all admit; it is shown by the crowding and irregularity of the teeth and the open bite. This crowding can be seen in early life, it is often demonstrable at four to five years of age. The "spacing" of the temporary teeth is often completely absent. It may quite well be that the moulding of the jaw has a stunting effect on its growth. This is not constant: some compressed jaws are apparently well developed, but the majority certainly show signs of more or less deficient growth. Appearances suggest that the want of growth is most marked in the pre-maxillary portion of the upper jaw, and I am convinced that in a large number of these cases congenital syphilis is present. I have repeatedly seen deficient development with an open bite, and sometimes an underhung bite, in cases of congenital syphilis without any adenoids or any lateral compression. In well-marked cases of congenital syphilis if the head is viewed in profile there may be seen not only a depressed bridge of the nose, but a sinking in of the whole nose and of the upper lip due essentially to a deficient development of the pre-maxillary portion of the maxilla. I incline to believe that congenital syphilis will be found in most cases where this ill-development of the pre-maxillary portion of the upper jaw is particularly marked.

That is really all I have to say in a constructive sense. To enumerate all the theories which have been brought forward to explain the conditions found, and to comment on them, to follow all the explanations of the way various forces, and in particular nasal obstruction, have been assumed to act would take up too much time. There are undoubtedly other factors at work which vitally influence the development of the jaws. Some of them at present are perhaps entirely unknown, others more or less suspected, such as the influence of the ductless glands, especially the thyroid and the pituitary, the influence of mastication and of various foodstuffs—branches of the subject recently discussed.

One further point I should like to raise, as I wish to put in a good word for the much-abused "baby's comforter." I am afraid I shall thereby lose whatever respect you may up to now have entertained for my opinions. I can only say that after some observation and inquiry I have never yet seen a single case in which the "comforter" has produced the deformities of the jaws which we have been discussing. I have known numerous babies who have used it regularly without the

least ill-effect. Whether it is clean or not depends entirely upon the care that is taken with it: there is no difficulty in keeping it clean, and it is no dirtier than most of the other things an infant puts in its mouth. As to its soothing influence upon the infant's nerves, I believe all those who are the parents of vociferous infants can, and I trust will, testify. My personal experience is that after enduring one infant for upwards of two months purely out of prejudice we gave way, and the result ever since, and with subsequent babies, has been peace. It is very regrettable if such a simple, harmless and useful appliance should be condemned by dental specialists without good cause. I trust you will consider this subject with open minds.

DISCUSSION.

Dr. SIM WALLACE said: We have listened to an excellent exposition of a slight modification of the true theory, if it is not simply a reversion to the theory of Sir John Tomes which was brought forward about fifty years ago. Sir John Tomes's theory was not universally accepted, probably because there are a large number of contracted arches—say 25 per cent.—where no history of mouth-breathing can be made out. The flaw in the theory which we have heard to-night results from want of recognition that the tongue has a large share in the stimulation of the development and growth of the mandible and of the maxillæ. It may help to illustrate the true theory if it is recollected that extirpation of the eyeball in infancy gives rise to lack of development of the surrounding orbital bones. We do not say that the bones round the orbit do not develop on account of the pressure of the orbital muscles. If the mouth is constantly held open, whether there is mouth-breathing or not, the developmental stimulus of the tongue which should habitually occupy the vault of the palate is absent and consequently the vault is not broadened and a stimulus to the development of the maxillæ is in abeyance. On the basis of this tongue theory also, deficient development of the jaws and crowding of the teeth are easy to explain, for anything which interferes with the growth of the tongue or anything which prevents it being kept more or less constantly in the vault of the palate correspondingly gives rise to deficient growth of the maxillæ. Thus chronic emaciation in childhood, associated as it is with corresponding emaciation of the tongue, is practically invariably associated with a certain lack of development of the jaws and crowding of the teeth. There are other factors which stimulate the growth of the tongue and muscles of mastication which, in their turn, tend to stimulate the normal development of the dental arches, but which need not be alluded to at present. Let me refer, however, to the two cases associated with facial paralysis on one side. Facial paralysis is not necessary for securing the regular arrangement of the teeth. When facial paralysis on one side plus mouth-breathing exists, then unequal pressure may exist on the two sides, the non-paralysed side may press most on the outer side of the arch, especially during the contraction of the facial muscles—e.g., during mastication. On the other or lingual side when the mouth is closed during mastication the tongue may be deflected to the paralysed side, and the dental arch on the paralysed side may thus be broadened as much as it would be under normal circumstances. Strictly speaking, it is not mouth-breathing which causes contracted arches, but something very frequently associated with it—i.e., lack of pressure of the tongue. The point has some slight practical importance, because more or less habitual open mouth without mouth-breathing is occasionally seen and the removal of adenoids does not always restore the habit of keeping the mouth shut. The moral of these remarks is that we should (1) prevent open mouth and mouth-

breathing by combating the pernicious practice of forcing children to sleep in winter in rooms which invite the pouring in of cold and damp air, and (2) we should undermine the present theory and practice of dietetics, which encourages lack of mastication and retards development of the muscles which hold the mouth shut, and leads to oral sepsis and chronic emaciation.

Mr. W. RUSHTON said that to him the most interesting part of Dr. Lack's paper was the case of the patient who had been paralysed on one side of the face. Dr. Lack's argument, as he understood it, was as follows: The patient, a mouth-breathing child, was paralysed on one side of the face; the muscles on the normal side, when the mouth was open, exerted pressure on the developing maxilla on that side and thus impaired its growth: on the paralysed side, however, the muscles did not exert pressure, and the maxilla on that side consequently was free to develop and did develop. Dr. Lack had brought the model as proof that his contention was correct, but to his (the speaker's) mind it was no proof at all. If any unprejudiced dentist accustomed to treating dental irregularities examined the model he would see that the development of the palate on each side of the median line was practically the same, but that the teeth on one side of the jaw were crowded while the teeth on the other side were not. The regular alignment of the latter was not due to the better development of the bone on that side—which was not the case—but was due to the fact that one of the premolars was absent, thus allowing the remaining teeth to attain to regular alignment. They must also bear in mind that, although the muscles on one side were paralysed, yet when the mouth was open they were put on the stretch. The whole subject was still very obscure and they must be careful not to accept alleged proofs without examining them very closely. He hoped Dr. Lack—whose paper they had all appreciated—would be able to secure the model and the data of the Glasgow case of which he spoke, and bring it before them at some future date. With regard to the causation of "open bite," he thought there were various factors, and that one was that the mandibular angle in the mouth-breather sometimes became rapidly very obtuse and the parallelism of the lower teeth to the upper ones was thus lost, the molars only touching.

Dr. WILLIAM HILL agreed with Dr. Lack that in mouth-breathers the pull of the dropped mandible when long continued during early childhood was probably a powerful factor in the lateral narrowing and arching of the hard palate. But he (the speaker) many years ago had written that the pressure of the tongue was another important factor. In normal nasal breathers with the mouth closed, the tongue filled the buccal cavity and pressed against and expanded the palate, exerting especially a continuous lateral tangential pressure along its borders; in mouth-breathers the tongue dropped as well as the lower jaw, and the latter was then free to exert a narrowing pull on the lateral alveolar borders. These mechanical explanations scarcely accounted for the actual stunting of the upper and lower jaws occasionally observed, and he did not think Dr. Sim Wallace's theory of what he called the absence of the

“developmental stimulus” exerted by the tongue when the mouth was closed led us much further, as it did not explain the stunting of the body generally which was marked in some adenoid children. He (Dr. Hill) thought these nutritional changes were due in part to respiratory insufficiency and were accompanied by early ossification, as in rickets, and led to the jaws retaining their infantile character to some extent. It could not be assumed, however, that adenoids were necessarily the cause of bony deformities of the palatal arch, as the association was only observed in a small proportion of adenoid cases, and, on the other hand, they might exist without adenoids. And again, it must be remembered that such deformities were not always acquired, for quite apart from cases of congenital syphilis, they were occasionally inborn—i.e., inherited—features, a number of the children of a family resembling one of their parents in this respect. In spite of all these conflicting facts he still thought, with Dr. Lack, that nasopharyngeal obstruction, with consequent mouth-breathing, was the main cause of the palatal deformity in the majority of cases met with.

Mr. J. G. TURNER said he was practically in entire agreement with Dr. Lack’s views. The outstanding feature was the subnormality and [moulding of the maxilla and some other bones of the upper facial skeleton. The mandible was not subnormal, though the dental arch was often compressed and the bone sometimes moulded. Otherwise the child was, as often as not, well grown. If the condition were due to failure of some “internal secretion,” there must be some selective action in the working of these secretions. Answering Mr. Rushton, he pointed out that, assuming the remnant of tooth on the paralysed side to be a second premolar, the inward position of the second incisor and the overlap of the first on the other side must have been present before the second premolar had reached its present state of destruction—that is, neither their malposition nor the regularity of the arch on the paralysed side was due to the space provided by the loss of the second premolar, and the consequent pushing over of the teeth on the sound side to the injured side, but antedated it. He had seen a similar case at Waddesley Bridge Asylum, Sheffield, but had, unfortunately, been unable to get models. He was hence the more inclined to accept Dr. Lack’s case.

Mr. T. B. LAYTON complained that all previous speakers had that night put forward theories without producing much evidence in support of them. He showed a slide of the cast of a mouth with a well-developed arch in a girl who had long been a mouth-breather.¹ He thought that Dr. Lambert Lack’s figure was not of a typical arch in a mouth-breather, but of one which would take some considerable amount of finding. He thought that the figures of sections were purely theoretical without any evidence to support them, and expressed his disbelief in the bands of which Dr. Lambert Lack spoke. He felt that both nasal and dental surgeons had too narrow a view as to what was

¹ The figure, with notes of the case, is published in the *Dental Record* for October, 1914.

the normal and what abnormal in palates and arches; the shape of the face varied within wide limits without being considered abnormal, and it was reasonable to suppose that the palate would do so also. He regretted that he had nothing constructive with which to follow this destructive criticism, and concluded by asking the Section to consider the greater part of a paper which he had already read before the British Society for the Study of Orthodontics.¹

Mr. J. F. O'MALLEY said that he had been acquainted with Dr. Lack's theory of the cause of this deformity for some years and had examined many cases which he considered gave some support to the view. He was also conversant with "the influence of the tongue" theory mentioned by Dr. Sim Wallace, and also the nutritional one (rachitic) alluded to by Dr. William Hill. In his opinion sufficient evidence could not be produced to establish any one of them, but he considered that we would be much nearer the truth of this vexed question by ascribing to all three a share in the changes which took place.

Dr. DAN MCKENZIE said that several years ago, in order to test the value of a series of statements made that the baby's comforter was productive of the high arched palate, he had investigated over 200 cases of children with and without adenoids. The conclusions to which he came were published in the "Central London Throat and Ear Hospital Reports," and definitely settled that there was no connexion whatever between the comforter and the deformities mentioned.

Mr. GEORGE NORTHCROFT was pleased to hear that Dr. Lambert Lack, in his closing remarks, conceded that the cause of the crowding in a certain percentage of arches might be due to causes other than nasal obstruction. Two cases of a brother and sister had recently passed through his hands, both of whom had such marked nasal obstruction as to render operation desirable at the age of 3. One case was perfectly normal at the age of 6½, the other at the age of 5½ showed marked crowding, complicated by post-normal occlusion. Roughly speaking, one would imagine that, in each case, the nasal obstruction had been operating on the two maxillæ for the same length of time, and this seemed to show that, at any rate, nasal obstruction did not always produce the evil results that were attributed to it, and would lead one to suppose that there was often a third underlying cause which produced both the nasal obstruction and the crowded arch. In both the cases referred to the children were at the present time normal breathers.

Mr. E. D. DAVIS said: In my own experience all cases of nasal obstruction, even in adults, are not accompanied by deformities of the jaws, and conversely, but more rarely, deformities said to be produced by this cause are not accompanied by nasal obstruction. To quote figures, from my private practice, of

¹ *Dental Record*, 1914, xxxiv, pp. 646-653, 667-671.

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patients above the age of 6, there were only eighteen cases with deformity of the jaws out of 136 which had been operated on for mouth-breathing and nasal obstruction. The majority of cases of deformity appear to be hereditary, and the deformities are exaggerated by nasal obstruction, which is a factor in its causation. The V-shaped narrow arch frequently accompanied by open bite may be caused by the position of the tongue in the mouth-breather. Ziem's classical experiment in which he obstructed one nostril of an animal for a considerable period and then found deformity of the jaws, is not supported by the only two adult cases which I have seen of unilateral congenital atresia of the posterior nares, in both of which the jaws were normal.

Dr. LACK, in reply, thanked the President and members of the Section for their kind appreciation and criticism of his paper. He had felt it was presumptuous to discuss dental subjects before their Section, but was very pleased with the helpful criticisms his paper had received. In answer to Mr. Turner, he would say that a deflected septum associated with a normal palate was probably traumatic in origin. Ziem's experiments on puppies did not agree with the results of the similar experiment carried out by Nature in the human body—namely, the condition of unilateral congenital choanal atresia in which, as noted in Dr. Lack's paper, and as confirmed by Mr. Davis's remarks, the effect produced on the palate was symmetrical, no matter whether the palate was deformed or whether it was normal.

Odontological Section.

February 22, 1915.

Mr. J. H. BADCOCK, Vice-President of the Section, in the Chair.

Some Primary Factors in the Causation of Gingivitis.

By R. ACKERLEY, M.B.

It is more and more being recognised that any septic condition of the mouth may have, and frequently has, a wide-reaching prejudicial effect on the general health, or on the health of one or other organ. It is, too, I think, the experience of others as well as my own that when oral sepsis has become chronic the difficulty of eradicating it is very great. It is therefore all the more important that we should inquire very carefully into the factors, especially the primary factors, which render the gums vulnerable to the pathogenic organisms which surround us so universally that escape from them is absolutely impossible.

If we except acute infections or traumatic influence, a lowered vitality of the gingival tissues is, I think, admitted to be a constant or almost constant factor in chronic gingivitis. On this point I should like to quote the following: (1) E. E. Talbot ("Interstitial Gingivitis," 1899, p. 163) writes: "Healthy gum tissue is intolerant of bacteria and will resist invasion of micro-organisms"; and again, "lowered vitality of tissue is a fruitful source of infection and suppuration." (2) In a discussion on a paper read by Mr. W. Hern at the Metropolitan Branch of the British Dental Association on June 17, 1914, the President of the Section is reported in the *British Dental Journal*, January 1, 1915, p. 15, to have said: "Again, with regard to the liability of the gum to infection, it was of prime importance to prevent congestion and consequent loss of tone. It seemed to him there was no mystery about the nature and cause of pyorrhœa. What was pyorrhœa? It was a chronic septic alveolitis. What were the organisms concerned? The ordinary micro-organisms of the mouth. Where did they first gain admittance? At the edge of the gum where it was really also the edge of the pericementum. Why did they succeed in thus invading a living

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tissue? Because the tissue was congested, lowered in vitality, lowered in resistance." Lastly (in "Stomatology in General Practice," 1913, p. 61), Pickerill writes of pyorrhœa: "The direct cause in all cases is infection by pathogenic organisms. It is now known that no single organism is responsible for the production of the disease, but that the infection is always an extremely mixed one. The organisms most frequently isolated from this disease are *Micrococcus catarrhalis*, streptococci, *Bacillus septus*, *Bacillus necrodentalis*, staphylococci, and the pneumococcus. These organisms are or may be, of course, present ordinarily in a healthy mouth, without giving rise to any local lesion. The factor which determines their entrance into the periodontal ligament is either an increase in their virulence or a decreased tissue resistance on the part of the patient. This latter may be brought about either through local or general causes." And the same author (in "The Prevention of Dental Caries and Oral Sepsis," 1912, p. 125) writes: "It is, too, a general pathological law, and not confined to the human tissues, but pervading the whole of organised Nature, *that stagnation in any tissue or cavity makes for decay and disease, and favours the development of those general analytical forces represented as a rule by the activity of micro-organisms*; and, conversely, all those forces which prevent stagnation and promote movement and circulation make for health and development."

If it is agreed that the lowered vitality of gum is a main predisposing cause of gingival disease, and if lowered vitality is mainly due to stagnation, we can proceed to inquire into the factors that lead to stagnation. Though other agencies may enter into causation, it is a priori probable that the factors will be found mainly in some faulty food habit, or in faulty food or drink.

As regards food, I am in general accord with those who lay stress on the abuse of saccharine and starchy foods, and am in agreement with the advice on those points given by Dr. Sim Wallace, Mr. Dowsett, Mr. J. G. Turner, Mr. Colyer, and many others. On this head I may be allowed to add two suggestions: (1) That not only is stone-ground wholemeal vastly superior to ordinary white flour, but that unleavened bread is not only more wholesome, more nutritious, and more palatable than leavened bread, and that it leaves far less residue on the teeth and gums. (2) That the addition of fat to white flour, whether especially in the form of pastry or hot buttered cakes or hot buttered toast or sandwiches containing tenacious fat, such as ham fat, leaves a slimy coating over the gums far more tenacious than that left by mere white bread or bread and butter.

As regards food habits, we are all agreed in theory as to the importance of thorough mastication, but in practice our standards would be found to differ very considerably. For the prevention of stagnation it is not only important that food—too often rendered unduly soft by the process of cooking—should be triturated, it is important that it should be sucked. If any of those present have had experience of eating ripe wheat out of the ear, or care to try the experiment of masticating fully some unground corn, they will find that they have to *suck* at the materials in their mouths and therefore pull on the cheeks and gums in a totally different and much greater degree than they would were they eating bread—even though the bread were in the form of crust or crisp toast; and this pull leads not merely to increased action of the salivary glands, but to more active circulation in the gingival structures.

But the factors to which I wish to direct your attention to-night are other than those that have so frequently been discussed, or I should not venture to address you this evening.

I do not know who first suggested mouth-breathing as a cause of gingivitis, as I find Talbot says: "Patterson holds that mouth-breathing has, in his experience, been a very common accompanying condition which he cannot help connecting with the production of the disease. By it the gums are kept dry, their functions destroyed, and the way paved for catarrhal inflammation" (Talbot, "Interstitial Gingivitis," p. 8). But, in any case, Colyer emphasises the importance of this factor and says (J. F. Colyer, "Dental Disease in its relation to General Medicine," 1911, p. 99): "It cannot be too strongly insisted on that mouth-breathing is the principal cause of gingivitis." But mouth-breathing is not a primary condition. The newborn child is not a mouth-breather; the condition comes on coincidentally with or subsequent to a general congestive condition of the nasopharynx, which in its most pronounced form shows itself as adenoids and enlarged tonsils. Careful observation for nearly thirty years has led me to believe that this congestive condition is due (1) to the habit of keeping the night nursery and living-room for children in all strata of society much too warm: the warm, moist atmosphere sets up an œdematous swelling of the mucous membrane of the nose and pharynx, and probably of the mouth, which has been well demonstrated by Professor Leonard Hill (*Lancet*, May 10, 1913, pp. 1288 *et seq.*); and (2) this congestion is further increased by the prevalent habit of overfeeding the infant and young child, which leads to chronic congestion of the coats of the stomach and of the nasopharynx and mouth, for, as Talbot puts it,

“there is direct communication with the gingival structures by blood-vessel and nerve throughout the mucous membrane from the nose, stomach, and lung.”

The rules generally laid down as to frequency of feeding the suckling and the young child are, I am convinced, in the direction of enjoining too frequent and, for bottle-fed infants, too copious feeding. It is impossible to enter into this detail here, but I may say, in passing, that the healthiest infant I have ever seen was one that was fed like a puppy in the first four weeks—i.e., only when food was desired. It was never suckled oftener than six times in twenty-four hours, and usually, even in the first four weeks, only five times, and later, when weaned, was fed at first only four times, and at the age of 19 months only three times in the day. These two factors—warm moist air which, besides causing congestion of the nasal and pharyngeal mucous membrane, is also far more laden with pathogenic organisms than cold dry air, and chronic congestion of the stomach from over-feeding—are, I suggest, primary factors in causing mouth-breathing.

But there is another food habit originating in the nursery which, I am going to suggest, is as injurious or even more so, especially in young adult and later life. Mr. J. G. Turner and others have pointed out that gingival disease, except as a result of trauma, is very rare in wild animals, comparatively rare in primitive man, and exceedingly prevalent in our own and other highly civilised, or shall I say artificialised, communities. Now quite apart from the facts that wild animals have natural foods, and live on the whole in the open air, they never get any food or drink hotter than the body—whereas in our own and other highly civilised communities the habit of taking food and drink hotter, and considerably hotter than, the body is general. It begins in the nursery. The breast-fed child escapes for a few months, but except in a few nurseries where the mother or nurse is more than ordinarily careful, and tests the temperature of the bottle with a thermometer, the bottle-fed child begins to have food hotter than its body a few days after birth. I have found a monthly nurse giving a young baby a bottle of food over 120 F., and being surprised that I found fault with it. She was quite sure that she could test correctly the temperature of a bottle by her hand or tongue. After the suckling stage we have the *hot* pappy foods—bread and milk, porridge, milk puddings, broths, hot tea or cocoa, hot potatoes, &c.—regarded as essential articles of food, so that before the child leaves the nursery a tolerance and even a liking for foods considerably hotter than the body has been established.

Pickerill ("Prevention of Dental Caries," p. 272) gives an analysis of the dietary of 1,500 public school children on a single day: and from that one sees that, apart from hot meat or hot puddings, at breakfast 53·8 per cent. took porridge presumably hot; 74·7 per cent. some form of hot drink—tea, coffee, or cocoa; at dinner, 46·2 per cent. took some hot drink; at tea, 76·6 per cent. took some hot drink; and in adult life hot food and drink are indulged by most of us at every meal. How hot one does not realise until one takes the trouble to test it by a thermometer; but many people take beverages well over 160° F., and food (potatoes, stews, &c.) at the same temperature; and soup at 120° F. to 130° F. is described by many people as being cold. Now let us consider the effect of this on the mouth, and especially on the gums, which necessarily come in contact with these hot foods. The frequent and prolonged application of heat to any part of the body, unless it is followed immediately by the application of cold, leads to a chronic congestion; and this habit of hot food is persisted in day after day, month after month and year after year.

In the last few years I have come across several patients with considerable chronic gingivitis who have not been mouth-breathers, have kept their teeth scrupulously clean and done all in the general way of oral hygiene that could be desired; but the gingivitis has persisted in spite of all care. These people are all, I find, in the habit of taking excessively hot drinks; and I have not one but many cases where the gingival condition has greatly improved when hot drinks have been given up. The worst case of gingivitis, with absolutely clean sound teeth, I have ever seen was in a young well-educated Arab who, in adopting English food, had also adopted the habit of taking freely hot tea and large cups of hot coffee, as opposed to the Arab habit of very small cups of coffee—not as a rule taken very hot—and even then frequently cold water is taken immediately before or after it.

I submit that besides errors in the character of food taken, the main factors in producing the lowered congestive condition of gum which renders it vulnerable to microbic infection are: (1) A chronic hyperæmia due to the habit of taking food and drink hotter than the body; (2) a chronic congestion of mouth, throat and nose due to hyperæmia of stomach, the result of over-feeding and too frequent feeding; and (3) the effect on nose, pharynx and mouth of warm, moist, unwholesome microbe-laden air; and that these factors should be regarded as primary in the causation of gingivitis.

(The diagrams illustrating Professor Leonard Hill's paper, "Physiology of Open-air Treatment," were thrown on the screen. These showed how in warm moist air the mucous membrane of the nose was swollen and congested, and how the congestion was relieved on a return to outside cold air.)

DISCUSSION.

Dr. SIM WALLACE said that he agreed with much that Dr. Ackerley had said, but was convinced that cold and damp air was much more productive of depressed vitality of the mucous membrane of the nasopharynx, and consequent liability to infection, than was warm moist air. He referred to certain statistics which appeared to show that "adenoids" were ten times more prevalent in children who were brought up under the strict open window night and day régime than they were in children who slept with the windows closed. He referred also to the fact that almost all young animals—for example, rabbits, squirrels, and mice—lived and slept in a warm, damp, ill-ventilated atmosphere, rather than in the cold damp current of air which was liable to be the lot of children who slept with the windows open in winter in this country.

Mr. J. G. TURNER thought the marginal gingivitis was a reaction to irritation due to germ infection and was a sign of vitality. It was not a predisposing cause of infection. Animals in the London Zoological Gardens suffered continuously from gingivitis, but got no hot food. He asked how Dr. Ackerley thought an infected stomach produced mouth hyperæmia—by reflex or by infection? He hoped he might be allowed to take exception to the words "scrupulously clean." In other parts of the body infection was conceded to be microscopic, and in the mouth the same must apply. Hence to allow the term "scrupulously clean" a microscopic examination must be made and found negative.

Mr. J. F. COLYER said he would like to correct a little misunderstanding that seemed to have arisen with regard to the statement he had made as to the relation of mouth-breathing to gingivitis. He had not stated that gingivitis was always due to mouth-breathing, for he was fully aware that there were many other causes; what he had laid stress upon was that a marginal gingivitis limited to the anterior teeth was always associated with mouth-breathing, the result of nasal obstruction.

Dr. ACKERLEY, replying to Dr. Sim Wallace, said that as he had never lived in a rabbit burrow he could not speak of the condition of its atmosphere from personal experience, but he knew it was not artificially heated. If Dr. Sim Wallace would produce diagrams which showed results of moist stagnant air different from those published by Professor Leonard Hill it would be more to the point. Mr. J. G. Turner regarded the congestion of gums antecedent to gingivitis as due to a reaction to micro-organisms. In this he differed from Talbot and other observers. Chronic congestion due to mechanical and physical causes was common, and he (Dr. Ackerley) contended

it was so in the mouth. He was in general agreement with Mr. Colver as regarded the evil effects of mouth-breathing, but his contention was that mouth-breathing was itself due to antecedent factors. He thought, too, there was a decided difference so far as the gums were concerned between mouth-breathing due to mechanical obstruction of nose and mouth-breathing due to a general congestive condition of the nasopharynx. As regards hot drinks, he had not expected that his views would be accepted that night. He had himself been watching the effect of hot food and drinks for many years, and he thought that his critics, when they had carried out prolonged observations on this point, would come to agree with him. There was a great danger in accepting a universal habit as a good habit; and it was not easy to trace the effect of a habit universal in the community in which one lived. But besides differences in the kinds of foods taken, one had to look for possible differences in food habits to account for the prevalence of gingivitis in highly civilised communities and its rarity among primitive men and wild animals.

The Influence of the Thyroid Gland upon Dental Caries.

By HERBERT EWAN WALLER, L.R.C.P.Lond., M.R.C.S.Eng.
(Birmingham).

DENTAL caries, though a well-worn subject, is still profitable for discussion. And I venture to state that the real cause is not yet appreciated in its true light. Without wishing to belittle the patient research and brilliant results of those who have demonstrated the essential influence of carbohydrate fermentation on the actual process of decay, I take this opportunity of protesting that it is unwarrantable, on that account, to assume that dental caries is merely a question of diet.

The details of the process of decay are well known to you, but to avoid any misunderstanding perhaps I ought to recite them. First, then, we have the lodgment of carbohydrate material upon the teeth. Secondly, the fermentation of this material, brought about by organisms in the mouth, with the consequent production of acids, of which lactic acid is the chief. Thirdly, solution of the lime salts of the enamel and dentine by these acids; and, finally, a further destructive process by bacteria, which liquefy the decalcified dentine. This I believe to be a fair summary of present opinion on the subject. Granting, then, that carbohydrate fermentation is an essential factor in dental caries, does it follow that increased dental caries is merely due to an increase of carbohydrate diet? I think not, for the following reasons:—

Firstly, we have the age-incidence. Dental caries is far more

prevalent, and more acute, in childhood and early adolescence than in maturer life. Is it really reasonable to explain this away on the assumption that the younger generations eat more sweets? After all, many grown-up people are very fond of chocolates; and, also, sweets form such a relatively small portion of the total daily intake of carbohydrates that the explanation is obviously weak. What about bread, toast, biscuits, potatoes, pastry, porridge, jams and marmalade, farinaceous puddings, and so forth, which form the major part of our daily rations? Is it likely that a small proportion of added chocolate or other sweetmeats would make such an important difference? Even if we insist that the offending carbohydrates must be sticky, and capable of clinging to the teeth, it is by no means self-evident that the diet of the younger generations differs much in this respect from that of their elders, whereas, on the contrary, the difference in the incidence of caries is very marked.

The second difficulty is the marked difference in the liability to caries shown by the various groups of teeth. For instance, the front teeth of the lower jaw, whether of first or second dentition, show a remarkable relative immunity from caries, when compared with the corresponding teeth of the maxilla. Why should starches and sugars cling to the upper teeth and not to the lower? By the laws of gravity the lower teeth would afford the more favourable resting place. Arguments that the lower teeth are better cleansed than the upper by lips and tongue are, at best, unconvincing. Yet there must be a reason for the undoubted difference in the incidence of caries. Again, if caries be merely a question of the facility with which carbohydrates may lodge on the teeth, surely the interstices *between* the teeth would invariably form the first point of attack, rather than the occluding surface. Yet caries more often commences on the occluding surface. Further reference will be made to these points later, when we shall see that the order of liability to caries is perfectly natural when considered in conjunction with our own view—that the varying alkalinity of the saliva is the dominating factor.

In the third group of difficulties must be placed the problem of arrested caries, and the influence of pregnancy and lactation in producing caries, and also the influence of general conditions of ill-health, such as anæmia and tuberculosis, and special conditions such as myxœdema, Graves's disease and diabetes. I think that if any uninitiated, but observant, general practitioner were asked to state the cause of dental caries he would reply, "It is partly a matter of general

health." And from the clinical point of view he would be perfectly correct. He would be quite familiar with the unhealthy anæmic type of patient who says, "Oh, yes, my teeth have all decayed because I have taken so much medicine." Of course, the position is untenable, because it is only the acid mixtures that would be chemically capable of attacking the teeth; and in view of Professor Pickerill's recent researches, which show clearly that vegetable acids provoke a free flow of alkaline saliva that would speedily neutralise any small quantity of acid that might remain in the mouth, it seems very doubtful whether an ounce of very dilute mineral acid taken thrice daily would have any material effect upon the teeth in the production of caries. But as no one, so far as I am aware, has attempted to maintain as a general thesis that dental caries is due to the ravages of mineral acids prescribed by medical men, we may dismiss the point. As to the influence of pregnancy. Many mothers have told me how they have lost their teeth during pregnancies; and "a tooth for every child" is an aphorism on the lips of every old midwife.

With regard to arrested caries, it will be sufficient for my purpose to give you a brief quotation from Smale and Colyer's "Diseases and Injuries of the Teeth." On p. 264 (second edition) we read: "Patients in whom this change takes place have nearly always undergone some marked improvement in health." And again, on the next page, "General improvement in health is, no doubt, an important factor, and by indirectly improving the local conditions existing in the mouth, prevents the development of acids and the growth of bacteria." No attempt is here made to explain the arrest of caries in a particular case mentioned, by any suggestions as to a change in his diet, although the man went for a sea voyage on account of his health. We are given the much more rational view that arrest of caries was due to general improvement in health. If we admit this in a case of arrested caries, there is obviously no reason why we should not at once admit the probability of the converse proposition and state that "Dental caries is due to a departure from normal health." Further, this is not only reasonable, but it is the only attitude we can adopt in view of the various difficulties that have been enumerated. Let us follow it up. We know the stages in the process of caries, and so far as we can ascertain, the enamel of fully erupted teeth is incapable of further modification by the ordinary nutrient fluids of the body, and apparently requires no nutriment, so that it probably cannot be affected from within. This view is confirmed by some recent experiments of

Professor Pickerill. Then, if caries be due to departure from normal health, the change must, obviously, occur in the fluids that bathe the teeth, that is to say, in the saliva, a suggestion more than hinted at in the reference we have just quoted concerning arrested caries. Our next step, therefore, is to see whether the saliva be capable of protecting the teeth from caries, and also whether there be any evidence that it actually does so.

The first part of the question is a simple chemical one. We have seen that the starting-point in dental caries is acid, produced by fermentation. But healthy saliva is alkaline, and should neutralise such acids at the moment of their birth, before they can attack the teeth. I have just said that saliva is alkaline. But if you test it with litmus paper you will frequently find that the parotid saliva is acid, though this is by no means always the case, and the submaxillary saliva is more often alkaline than not. This acidity or alkalinity is merely a question of the proportion of alkaline salts present, and is therefore to some extent a quantitative test. Blue litmus paper, being sensitive to carbonic acid, will turn red if this gas be present in excess, or, differently expressed, if the quantity of alkaline salts present be insufficient to let it remain blue. And the proportion of calcium bicarbonate and carbonic acid gas dissolved in the saliva, even in the case of the feebly alkaline parotid saliva, ought to be such that the red litmus paper turns faintly blue. I mention these details because objection has sometimes been raised to my use of litmus paper as a test for the saliva, on account of the fact that it is so easily reddened by CO_2 . The fault, however, is not in the litmus paper, which is really a test of extreme delicacy, but in the defective saliva of our hyper-civilised race, in which, consequently, it is very hard to find a mouth that has escaped caries. The submaxillary saliva has much greater reserves of alkaline salts than the parotid, and in consequence frequently shows the alkaline reaction. The chemistry of the saliva is aptly illustrated by that of soda-water, which is worth considering.

The name soda-water suggests alkalinity, and bicarbonate of soda is usually present, to the extent of 10 gr. in a pint, though formerly the quantity used to be 30 gr. Yet soda-water is acid owing to the carbonic acid gas which is dissolved in it under pressure. But if the gas be driven off by boiling, or even be allowed to evaporate slowly, the remaining fluid becomes alkaline to litmus paper, owing to the bicarbonate of soda which is still present in solution. And the addition of 1 or 2 gr. of bicarbonate of soda to a test-tube full of soda-

water produces a strongly alkaline fluid. Now, similarly, the saliva is essentially a solution of calcium bicarbonate and carbonic acid gas in water. But the carbonic acid gas is not under pressure, and the proportion of calcium bicarbonate to CO_2 is such that in health the saliva is alkaline, and when it proves to be acid, it is perfectly reasonable to assume that such acidity indicates a deficiency of alkaline salts, seeing that there is no possibility of great variation in the small amount of CO_2 normally present. The alkalinity of the parotid saliva is due entirely to calcium bicarbonate. The sublingual saliva contains also calcium phosphate, and the submaxillary saliva contains carbonate and phosphate of magnesium as well as calcium. Now, phosphoric acid is a tri-basic acid, and there are, consequently, three different phosphates of calcium. One of these, that in which the whole of the hydrogen of phosphoric acid is replaced by calcium, could only exist as a solid, and is not, therefore, present in the saliva. The other two salts would contain respectively one and two molecules of calcium and would accordingly be acid in the former case and alkaline in the latter. But, again, the acid phosphate of calcium could not exist in the presence of any carbonate, as it would speedily attack the latter, with production of CO_2 , and would itself become alkaline. And as calcium bicarbonate is normally present, it follows that the calcium phosphate present must be the alkaline phosphate, that is, the di-calcium monohydrogen phosphate. The advantage of this alkaline phosphate is obvious, because in time of need one of the calcium molecules could be sacrificed towards neutralising any free acid in the mouth.¹

If, then, we may ignore the magnesium for a moment, it becomes obvious that the alkalinity of the saliva depends on the amount of available calcium: and seeing that alkalinity is the essential chemical factor in protecting the teeth from caries, it follows that this protection is most likely to fail when there is a relative shortage of calcium, which is the case; for dental caries is most prevalent in childhood and youth, when relatively enormous quantities of calcium, and also a small amount of magnesium, are required for building up the skeleton. The same argument applies to the conditions of pregnancy and lactation, when the maternal supplies of calcium and magnesium are required for the bones of the fœtus and for the production of milk. Magnesium metabolism is possibly not a factor of great importance in connexion with dental caries, but we may remark that the phosphate and

¹ The three phosphates of calcium are: $\text{Ca}_3(\text{PO}_4)_2$, $\text{Ca}_2\text{H}_2(\text{PO}_4)_2$, $\text{CaH}_4(\text{PO}_4)_2$. The interaction between the acid phosphate and calcium bicarbonate is represented thus: $\text{CaH}_4(\text{PO}_4)_2 + \text{CaH}_2(\text{CO}_3)_2 = \text{Ca}_2\text{H}_2(\text{PO}_4)_2 + 2\text{CO}_2 + 2\text{H}_2\text{O}$.

carbonate of magnesium, which are present in the submaxillary saliva, would have a chemical effect similar to that of calcium in neutralising acids, and so preventing the incidence of caries. This fact must help to turn the scale in favour of the preservation of the lower front teeth which we have already mentioned. To some extent the metabolism of magnesium throughout the body follows the same lines as that of calcium. Bone contains nearly sixty times as much calcium as magnesium, but the amount of magnesium in the urine is about four-fifths that of calcium (Foster's "Physiology"), and the two are usually dismissed in an ignominious group as "earthy phosphates." The said earthy phosphates are also excreted in the fæces, crystals of ammonium-magnesium phosphates being very conspicuous, and lime and magnesia soaps are also present. It is, perhaps, worth remarking the exactly opposite effects of calcium and magnesium upon the intestine. We are heedless of the metabolism of either when we freely prescribe chalk for diarrhœa and magnesium salts for constipation. I have read, however, that the ingestion of magnesium salts promotes the excretion of calcium, and similarly that the ingestion of sodium salts promotes the loss of potassium. Obviously the balance of alkaline salts in the body must be regulated in some way. That magnesium salts possess properties other than aperient has been shown in late years by the use of magnesium sulphate as an external application for erysipelas, for which purpose I have found it valuable on three occasions, and also by the use of intradural injections of magnesium sulphate as a remedy for tetanus. Finally, it is quite certain that magnesium would not be a constant ingredient of the saliva unless it served some important purpose.

We may now consider the relative incidence of caries in different teeth, and we shall find here the most powerful arguments for believing that the saliva is the dominating factor in dental caries. Why is it that the lower front teeth escape caries till every other tooth has perished? My own opinion is that this is simply and solely due to the fact that they are constantly bathed in saliva, which naturally flows to the front of the mouth when the head is in its ordinary position. The facts cannot be denied and the explanation is in no way abstruse or difficult. On the other hand, it is extremely difficult to support any other theory as to why these particular teeth should commonly be singled out for preservation from decay. And these very teeth are the most prone to become coated with tartar, which consists of lime salts deposited from the saliva. It is further interesting to note that the first lower bicuspid shows a somewhat smaller liability to caries than

the second bicuspid, in spite of the fact that it erupts earlier. The reason, I think, is simply because it is nearer to the perpetual bath of saliva enjoyed by the lower incisors and canines. The whole of the saliva secreted by the different salivary glands would gravitate to this part of the mouth. But so far as alkalinity is concerned, the parotid saliva is of much less value than that secreted by the submaxillary and sublingual glands. Hence, if there be any dearth of calcium salts this will first be felt by the parotid, just as the results of famine are first felt by the poor. And in that case we should expect the teeth normally under the protection of the parotid saliva to be the first to suffer. And this is precisely what happens. In young children the temporary molars are the first teeth to suffer from caries, and in older children the sixes and to a distinctly less extent the sevens. The period of growth is naturally a most hazardous time for the teeth, if a shortage of calcium in the saliva carries with it a risk of caries. For between the ages of 7 and 18—that is, roughly, from the commencement of the second dentition to the completion of the period of *rapid* growth of the skeleton—we must reckon that there are eleven years in which the saliva is liable to be stinted of calcium, whilst the skeleton is growing approximately 21 in. in the case of the male and 19 in. in the female, which involves a heavy drain on the calcium content of the blood. It follows that those teeth which are deprived of adequate salivary protection for the longest period are likely to suffer most from caries, if all other factors were eliminated. The only other factor that is invariably present is the tendency to lodge carbohydrate particles, which will necessarily vary in different mouths, according to various irregularities and so forth. This is probably the factor which causes the lower molars to decay more than the upper, but otherwise its influence is less obvious than would be the case if it were all-important. It is surprising how often irregularly placed teeth remain sound, in spite of the fact that every possible facility is present for the lodgment of carbohydrate in the immediate neighbourhood. And the premolars ought to decay infinitely more often than the upper incisors, for similar reasons. Yet such is not the case. There is clearly some other factor more potent than the mere facility for lodging carbohydrate. And if we consider the protective power of the alkaline saliva, coupled with the fact that this power is more likely to fail during the period of rapid growth, we shall be able to give a very reasonable explanation of the most common incidents of caries. We have already said that the lower front teeth, being constantly bathed in saliva are thereby protected from

caries. The lower canines share this protection and the first bicuspid are protected to a slightly greater extent than the second. The first molars are subjected to great stress, because they are the first permanent teeth to erupt during the growing period, and they have to stand the risks of the eleven years from 7 to 18, as already explained. The lower sixes erupt earlier than the upper, and by the laws of gravity are more liable to lodge particles of food. Hence they suffer even more than the upper sixes. The second molars erupt shortly before puberty, a period when growth is also very rapid, and they very often succumb to the risks of six dangerous years. Their position at the back of the mouth and the obvious facilities they offer for carbohydrate particles must also tend to their undoing, and again, the lower are exposed to greater risks than the upper. The bicuspid teeth erupt two years or more before these second molars, and on that account run a greater risk of caries. But this is to some extent counterbalanced by their position in the mouth, and the smaller surface which they present for attack. The canines cannot, on account of their shape, be fairly compared with any teeth but the incisors, which erupt four or five years earlier and are therefore exposed to much greater risks. The wisdom teeth erupt towards the end of the growing period, and have a consequent advantage, which is more or less counterbalanced by their unfavourable position in the mouth. I cannot say which factor is the more potent in this case, but I am inclined to think that the influence of late eruption is predominant. Such, I think, are the general principles governing the special risks of caries to individual teeth.

We must now deal with various exceptions and apparent incongruities. It is interesting to note that in later life the lower front teeth may be attacked by caries, after having outlived the extraction of all the other teeth. This, you may say, disproves what I have said about the salivary bath. I must take leave to differ. It gives strong support to my theory. You have extracted the molars and the bicuspid, and the alveolar margin has shrunken away. In short, you have drained my bath by sinking a hollow on each side of it, into which the saliva is bound to flow. In consequence the teeth are robbed to a great extent of their protection, and will in due course decay. And you will often find the caries on the apex, which is not the place where one would expect to find most carbohydrate lodged, though it is the part which will first lose its natural protection, as the level of the salivary bath is lowered. This danger will evidently be less where a denture is worn, because the latter will to some extent fill up the

hollows which otherwise drain the bath. Thus, were it not for other evils involved by wearing a denture, its presence would tend to prolong the life of the lower fronts, though it must be admitted that these commonly succumb to the ravages of pyorrhœa rather than to caries. One sometimes sees caries in the lower front teeth of young children. There is a probability that this may sometimes be the result of early extraction of the temporary molars, which would disturb the level of the salivary bath in a similar manner to that just described. And there is also another reason. To the little child most objects of interest are considerably above his own level. His parents and his nurse tower above him, and as he looks up continually with childish wonder at all the strange things around him, the saliva flows towards the back of the mouth instead of to the front. In a grown-up patient a carious lower incisor or canine should always excite some interest, and where the explanation is not obvious on the lines I have suggested, it is worth considering the bite, to see whether there be any upward tilt of the lower jaw. Also the peculiar carriage of which "Little Johnnie Head-in-air" was the prototype is occasionally to be seen among adults.

We shall comprehend some of the other anomalies of caries by the light of Professor Pickerill's researches on the influence of the saliva upon the teeth. His results are of special interest to me because they entirely confirm the conclusion at which we have already arrived—namely, that the saliva is really the dominating factor in dental caries. But he takes us a little further than I have done, because he shows that a period of good alkaline saliva, rich in calcium, may to some extent protect the teeth from lean years to follow. Concerning his experiments I shall quote from my paper read before the Wessex Branch of the British Dental Association in May, 1913, which was published in the *British Dental Journal* for July, 1913:—

"The teeth of Europeans may be divided broadly into two classes, malacotic and sclerotic, that is, soft and hard. These two varieties differ from each other in three essentials. The enamel of the malacotic tooth is physically softer, is more permeable by a solution of silver nitrate, which can be used as a stain, and is more readily soluble in lactic and other acids than is the case with the enamel of the sclerotic tooth. Further, an unerupted tooth is the most vulnerable in these points, but after eruption becomes less malacotic and more sclerotic. The same facts hold good in the case of the Maoris, a race peculiarly immune to caries, but with this difference—it was found in the case of a Maori that a tooth took but three years to acquire a degree of sclerosis that was only reached in eleven years by a European. An actual difference in the density of the enamel itself was found in these varieties of teeth. Evidently,

then, a change occurs in the enamel of teeth after their eruption, and this change occurs far more rapidly in the teeth of those races immune to caries than in the case of Europeans. Professor Pickerill's explanation of the change is that it is due to dialysis of the calcium salts of the saliva through Nasmyth's membrane, and deposition of these salts among all the minute interstices of the enamel, the deposition of the salts being caused by evaporation of the CO₂ which holds them in solution."

The experiments by which these conclusions are reached need not here be detailed; they are fully described in Professor Pickerill's book and appear convincing enough. And we may reasonably argue that the greater the available quantity of calcium in the saliva the faster the teeth would become sclerotic. It is also probable that these deposits of calcium salts in the enamel would be first attacked in the presence of any acids, rather than the enamel itself. Now these results of Professor Pickerill's will readily explain certain cases which might otherwise have proved difficult, because not in harmony with the general scheme of caries that I have already sketched. It must not infrequently happen that a child, aged 13, who has escaped caries in her first permanent molars may fall ill. The calcium metabolism is upset and the protection of the saliva becomes inadequate. The newly erupted second molars are now in much greater danger of becoming carious than the first molars which have reached a certain degree of sclerosis during the seven years since their eruption. Similarly, in the case of a child aged 11, the greatest liability to caries would be in the bicuspid, which erupt respectively in the ninth and tenth years. There is one other point worth mentioning here. Caries is not always symmetrical; but seeing that children spend, or should spend, nearly half of their lives in bed, it follows that a child who habitually sleeps on one side will unwittingly confer an advantage upon the teeth on the side of the face that is next the pillow, because the saliva will naturally flow towards that side. In the case of children who sleep with the mouth open the saliva has an unpleasant habit of flowing out of the mouth on to the pillow, and none of the teeth will benefit by its presence during the night. If children with adenoids really suffer more from caries than other children this is a possible factor, though I think that such children also suffer from an unstable calcium equilibrium. There seems to be little doubt that ill-health disturbs the calcium equilibrium, presumably via the ductless glands, and thus leaves its record on the teeth. Careful observation should enable us to read a good deal of the past history of our patients from their teeth, and Sherlock Holmes should have done very well in this line, if the idea had occurred to him. But many people

whose teeth are carious will not admit that their health has been bad. The deviation from the normal has been slight, and with the many aids of civilisation it has hardly been noticed.

We must now face another difficulty. We know that, as a rule, the teeth of various native races are remarkably good, whilst the teeth of civilised races are usually the reverse. We have traced the apparent influence of calcium metabolism on the incidence of caries in our own race. But you may well answer me that savage races also pass through periods of growth, pregnancy and lactation, and also of ill-health. Why is it that their calcium supplies are adequate to meet the strain, whilst ours are not? The question has possibly not been asked in quite the same form before, because calcium metabolism has not hitherto been given the premier place, which I hope, in the fullness of time, it will receive.

If we were to follow all previous precedents, the answer to our question would be looked for in diet. In former days it was the liability of diet to stick to the teeth and ferment. More recently it has been the power of diet to provoke an adequate supply of saliva. And I have sought for an answer in the calcium content of the food. Now I want to cast diet to the winds, at any rate as the essential cause of dental caries. Consider: The Esquimaux consumes perhaps a dozen pounds of meat a day, and has excellent teeth. The Mohammedan lives chiefly on rice and has excellent teeth. The Fiji Islanders and the New Zealanders, both cannibal races, have excellent teeth. Apparently it matters not what the uncivilised races eat, or how diverse their diet. Their teeth are invariably superior to the standard of the average European. And if the answer to the riddle were to be found in so simple a matter as the intake of calcium, we should expect to see a marked difference in the teeth of persons living in a hard water district. There is a slight difference, it is true, but only slight. Again, our young people consume a very large amount of cow's milk. And the amount of calcium and also that of phosphate in cow's milk is very rich, nearly six times as much as in human milk. Calcium is normally excreted in the urine to a definite extent, the daily output for a man, according to Professor Stirling, being 0.26 gm. I have tested a good many urines for calcium, precipitating the calcium as oxalate and centrifugalising the deposit in a graduated tube. The graduations in my tube have not been sufficiently delicate for me to record the slighter variations, and with few exceptions I have not met with gross variation. I may, however, mention a single case, that of an overgrown boy, aged

9 years 10 months. His height was 4 in. above the normal, and his weight corresponded with his height. His teeth were perfect, with the exception of four carious first molars. Though he was under 10 years of age his bicuspid and second canines were all fully erupted and the second permanent molars almost so. Examination of two samples of urine, both of rather low specific gravity (1014), showed a calcium content about double the normal. Here, then, we have a boy, eating ordinary food, living in a city with a soft water supply, assimilating calcium at a far greater rate than the normal, and yet excreting a large excess in his urine. This does not look as though there were any deficiency of calcium in ordinary food. The condition of his teeth is not open to argument, because one would not in the ordinary course of events expect to find caries in any of the permanent teeth in a boy aged 10, with the exception of the first permanent molars, where it had already occurred. But I should fear that caries is likely to follow in other teeth, in due course, if one is to judge from the present rate of calcium excretion. The boy is extremely nervous, walks in his sleep, and suffers slightly from functional albuminuria. Without pressing the fact that calcium is a powerful nerve sedative, as shown by its influence in epilepsy and also in tetany, we know that functional albuminuria is sometimes considered to be a sign of calcium deficiency. The error of calcium metabolism which is responsible for dental caries is not necessarily a very gross one. And it is really very wonderful that calcium should be absorbed at a relatively enormous rate for a certain number of years, during the process of growth, and that almost suddenly, when growth is complete, the rapid assimilation of calcium should be discontinued, without any special change in diet. Unless used for some other purpose the calcium must now be excreted, or else simply pass out of the intestine unabsorbed.

Reviewing all the facts, it is difficult to think that there can at any time be an actual deficiency, in the diet, of so plentiful an element as calcium. The fault, then, must lie in its distribution. We might, of course, inculcate the parotid gland, which is more or less the line taken by Professor Pickerill. The parotid saliva is undoubtedly the weak point, so far as dental caries is concerned, and Professor Pickerill blames our diet for this, on the ground that it is too insipid and alkaline to provoke an adequate supply of saliva. In short, that the salivary glands suffer from lack of stimulation, and that the cause is faulty diet. But we have already shown that diet will not meet the difficulties of special liability to caries in youth, in pregnancy, after illness, and so forth.

It is possible, however, that the parotid may be inculpated for other reasons. There is some evidence that it works in sympathy with the sexual glands. That evidence is exemplified by metastatic inflammation of the testicles, ovaries, and breasts in cases of mumps, and there is also a good deal of other evidence which I have discussed elsewhere and need not again mention now. We cannot, of course, dissociate the parotid from the other salivary glands, and we may note that profuse salivation is sometimes a feature of Graves's disease, and may also be witnessed in the opposite condition of thyroid deficiency. In these cases, however, it may depend on concomitant circumstances. It is certainly sometimes the result of treatment with iodine.

But we must return to the subject of calcium metabolism, for in this, it seems, we must look for the key to our puzzle. It is now agreed that calcium metabolism is directly controlled by the ductless glands. The place of chief importance has probably been hitherto awarded to the thyroid gland. The results obtained by thyroid therapy have been so vastly superior to other triumphs of medicine that one was perhaps inclined to lose sight of the other controlling influences. And it was on this account that, over five years ago, I made my first attempts to attribute the incidence of dental caries to the thyroid gland, relying on certain undeniable facts—namely, that the teeth are particularly bad in cretinism and in myxœdema, and in various other states which are amenable to thyroid treatment, and that their state might therefore be supposed to depend on thyroid deficiency. With increasing knowledge one is naturally enabled to re-arrange the facts and make better attempts at their interpretation. And I believe that I am now on solid rock when I say that dental caries depends upon poverty of calcium salts in the saliva, and that the commonest cause of this poverty is not deficiency of calcium in the food, but excessive excretion of calcium from the system, owing to disturbance of some one or more of the ductless glands. But it is still very difficult to say which of the internal secretions should have the prior place, because, as I believe, they are one and all mutually interdependent. If one is disturbed, others are thrown off their balance.

With reference to calcium metabolism we may reasonably consider the internal secretions from a threefold point of view, which is in accordance with the great scheme of Nature, as seen in both the animal and vegetable kingdoms. Nature's threefold cycle consists of growth, reproduction, and decay. Growth involves enormous assimilation of calcium. The active glands in this stage are certainly the thyroid and

thymus, probably the pituitary and suprarenals, and possibly the sexual glands. I do not say that the influence of yet other glands is without importance, but we have mentioned enough for the present. The calcium-building power of the thyroid gland is proved by the effect of thyroid treatment upon young cretins. That of the thymus rests on certain experimental evidence, a condition resembling rickets having been produced in some animals by ablation of the thymus gland. The influence of the pituitary gland as a calcium retainer is shown by the conditions of giantism and acromegaly, which are believed to depend on increased activity of that organ, and clinically there seems to be some evidence that pituitary gland substance checks the excretion of calcium. That the suprarenal glands are most potent calcium-builders is proved by enormous overgrowth in certain cases of suprarenal tumour. A case has been recorded in which "a girl, aged 7, was as tall as a woman aged 20." We need not, for the time being, consider the somewhat controversial question of the parathyroids.

It is difficult to evade the conclusion that the glands we have just mentioned are working together harmoniously during the period of growth to build up a healthy frame suitable for Nature's great purpose—namely, that of reproduction. This being accomplished, a remarkable change takes place. The thymus gland now atrophies, and instead of it we are provided with the fully matured sexual glands. And experiment has shown that these diverse organs are in some way complementary. If the immature testes be removed from certain animals, the thymus fails to atrophy, or even becomes enlarged. And if, on the other hand, the thymus be removed, the testes enlarge at an earlier date. Further, the normal atrophy of the thymus can be hastened by breeding from young animals. No influence on the rate of growth was noted by Noël Paton in the case of young guinea-pigs, in which either thymus or testes were removed, but removal of both sets of organs simultaneously caused a marked delay in the growth. We must, therefore, possibly accord to the testes, even before they are mature, a share in the skeletal growth. It must be remembered, however, that the testes often contain accessory bodies of the nature of the suprarenal gland, and, as we have already noted, the suprarenals have a great influence on growth. But now that the frame is mature, that is to say, has reached the reproductive age, after a longer or shorter period of relatively extremely rapid calcium assimilation, it becomes perfectly obvious that this rapid assimilation of calcium must be checked, or it would naturally become inconvenient. We know that the thymus is now cast aside, and that fact in itself is

suggestive. We also know that the ovaries assist in the elimination of calcium. Calcium is to some extent excreted in the menstrual fluid, and also, according to Blair Bell, removal of the ovaries lessens the excretion of calcium in the urine. The control over calcium possessed by the ovaries is convenient, because during pregnancy, when calcium is required for other purposes, it can be, and, as we believe actually is, conserved by cessation of ovarian activity. By analogy, it is probable that sexual activity on the part of the male produces increased calcium elimination. But it is also unlikely that Nature would rely entirely upon so intermittent or periodical an occurrence as sexual activity to reduce the daily accumulation of calcium. And in consequence I believe that this work is relegated to the thyroid gland. This supposition is supported by the remarkable sympathy that exists between the thyroid and sexual glands, which is more particularly noticeable in the female. Enlargement of the thyroid is prone to occur at the menstrual periods, as a result of sexual excitement, and also during pregnancy. As to the precise significance of this enlargement it is not easy to be dogmatic. It is usually argued that enlargement means hypertrophy in response to increased demands; but in the case of the thyroid I am quite sure that enlargement must sometimes be interpreted in the opposite way, being due to simple distension of the vesicles with colloid material which is not at the moment required, and is consequently being stored for future use. This process may sometimes be witnessed in cases of Graves's disease under treatment with iodine. For the time being, the artificially supplied iodine is adequate for the needs of the body, and the thyroid secretion is stored up instead of being discharged at the usual disastrous rate. This is proved by the immediate amelioration of the symptoms, concurrently with enlargement and hardening of the goitre. But if the remedy be now left off it not infrequently happens that the goitre suddenly diminishes in size, with an immediate and severe exacerbation of all the Graves's disease symptoms. So it is quite possible that the enlargement of the thyroid which occurs under sexual excitement, or at the periods, is due to reflex inhibition of the thyroid, owing to increased activity of the ovaries. The suggestion is supported by the fact that after ovariectomy, or at the menopause, we have, at first, distinct evidence of increased thyroid activity in the form of palpitation, flushings, tremors, insomnia, increased nervousness, and so forth. It is evident, then, that the thyroid and sexual glands are to a considerable extent complementary.

It is not easy to say what arrangements have been ordained for the

regulation of calcium metabolism in the waning years of life. Doubtless, in part, it is regulated by diminished appetite and lessened intake. To some extent it is probably still regulated by such vitality as remains to the thyroid and also the sexual glands. In this connexion it is worth noting that old people who marry again in later life not infrequently seem to regain some measure of juvenility. And Arnold Lorand, in his recent work, "Old Age Deferred," although himself a bachelor, advises re-marriage as one of the means of prolonging life. He appears to attach the greatest influence to the thyroid. On the other hand, the experiments of Brown-Séguard with testicular extract in his own old age are now almost monumental. And though there have been many scoffers we cannot altogether ignore his conclusions, for we have an independent authority in the testimony of Dr. Sajous, who saw the old man both before and after his experiments, and gives the verdict that he "literally looked twenty years younger." But the actual failure of calcium metabolism is no small factor in senile decay. In aged people the bones contain a larger proportion of calcium salts, and are consequently more brittle and liable to fracture. Calcareous deposits are prone to occur in various places, and death is often ushered in as the result of a rupture of some long calcified cerebral artery.

Such, then, is an imperfect and incomplete history of calcium metabolism. And dental caries, as we have said, is due to deficiency of calcium salts in the saliva. It is therefore of special interest to note the recently published experiments of Dr. Broderick. He estimated the calcium content of the saliva, and also the alkalinity, in some young children suffering from measles and from scarlet fever. He then administered small doses of a tablet containing thyroid, suprarenal and pituitary gland extract. At the end of two days of this treatment the alkalinity of the saliva showed a most remarkable increase. He was then so unfortunate as to contract scarlet fever himself. He carried out similar experiments with his own saliva and obtained entirely analogous results. The details are published in his most excellent paper in the *British Dental Journal* for January 15 of this year. I think that these experiments, coupled with my own previous observation that the alkalinity of the saliva in children may sometimes be restored by thyroid treatment, are absolute proof that the saliva is, in fact, regulated by the internal secretions. The precise part played by the thyroid is difficult to determine, and probably varies, both according to the age of the patient and also according to the activity of the thyroid. We know, from the example of cretinism, that adequate

calcium assimilation cannot proceed without due thyroid activity. And we know from experiment, and also from observations of Graves's disease, that excessive thyroid activity produces increased elimination of calcium. Possibly children require greater thyroid activity than adults, and again it is possible that so far as calcium metabolism is concerned the product of thyroid and thymus may differ from that of thyroid plus sexual glands. But in any case, since dental caries often occurs without any particular variation in the calcium intake, we must suppose that the deficiency arises from one of two causes, either defective assimilation or excessive excretion. This, of course, involves the whole question of the inter-relationship of the ductless glands. Are the thyroid, the pituitary, and the suprarenal glands over-fatigued and unable to absorb the daily ration of calcium supplied to them? That is not very likely, because if there were any serious exhaustion of such highly important glands the health or even the life of the patient would be seriously imperilled, whereas teeth decay even when one is not ill, in the ordinary sense of the term. It is more likely, therefore, that the deficiency of calcium is due to waste. Nature has kept the body alive, and in comparatively good working order. She has resisted or successfully overcome the inroads of disease, without any special inconvenience to the individual. But, nevertheless, there has been a little wear and tear. Some calcium has been lost in the process. That sounds much more likely, and is also in accordance with the observation that under the influence of excessive thyroid activity the excretion of calcium is much increased. We also believe that the thyroid gland participates in the protection of the body against disease, and that many of the symptoms which we group together under the term "fever" may actually be produced artificially by administering thyroid gland substance. It matters little for the moment whether these results be produced directly by the activity of the thyroid gland, or indirectly through the agency of other glands which are themselves influenced by thyroid activity. The point is, that excessive thyroid activity is capable of producing increased elimination of calcium, and so promoting those changes in the saliva which, as we have seen, are conducive to dental caries. So far as my own observations go, the teeth are usually very bad in Graves's disease, and in many cases of goitre. According to Dr. Hertoghe they are also very bad in myxœdema. But myxœdema is the result of thyroid exhaustion, and exhaustion is the result of previous activity. That activity need not necessarily have been so great as to amount to Graves's disease, for Graves's disease implies the

possession of a remarkably powerful thyroid gland. The more powerful the organ the less likely is it to become exhausted. So although Graves's disease is sometimes the forerunner of myxœdema, it is likely that myxœdema will more often result from the exhaustion of a much less powerful thyroid gland, consequent upon a period of over-activity.

Let us now look at our problem again. We are looking for a factor which is likely to produce dental caries by causing a wastage of calcium salts. And that factor must be strikingly operative under conditions of civilisation, and equally strikingly absent from the conditions of life among the savage and untutored races. What, then, are the essential differences between the civilised and savage races which could possibly provide the clue for which we are seeking? Diet we have discussed, and I have given my reasons for rejecting it. Clothing can only be considered from the point of view of warmth. And as the savage races of the Arctic regions and of the Tropics both enjoy comparative freedom from caries, we cannot make any use of that suggestion. Any considerations of temperance or morality should show an advantage to the civilised races. So should the considerations of health, hygiene, and cleanliness, with but one exception. Medical science is helping to preserve the unhealthy, and the unfit are able to reproduce their kind under conditions of civilisation, whilst among the savage races illness so commonly means death, which is undoubtedly a most effective way of preventing dental caries. That factor must not be ignored. But the cardinal difference between civilised and uncivilised races is that civilisation aims at cultivation of the brain, as opposed to purely physical existence. This process is yearly becoming more complex, and the struggle for existence in civilised countries is mental rather than physical. And this, I think, is where the thyroid factor comes in. You know that the untreated cretin is an idiot. In other words, the brain is unable to develop without adequate thyroid secretion. You are aware that the mental capacity of a myxœdematous person is decidedly below the normal. His ideation is slow, he suffers from a partial or entire loss of memory, and a difficulty in expressing himself. Contrast this general slowness, both of movement and mentality, with the general alertness of the successful man of to-day. And contrast it also with the natural disposition of the individual suffering from Graves's disease. The latter is well described by Dr. Leonard Williams in the *Practitioner*, January, 1915. He says:—

“ A sense of restlessness, both physical and mental, is characteristic and constant even in early and slight cases. But that is not all. Whatever she

may previously have been, the patient now becomes irritable, assertive, and wilful, unreasonable, wrong-headed, and 'difficile' to the last degree of impossibility. To make matters worse, the intelligence is not only unimpaired, but it is often hyperacute. The fondness for music, the drama, for artists, actors, and tenors is a pronounced and often a pathetic feature."

I have such a case now under treatment who used to enter and leave my consulting room like a whirlwind. I have many times urged her to go slowly, and she now sometimes succeeds. Of course, the advanced cases are often physically incapable of this tempestuous movement. Dr. Leonard Williams has described the female, but I have seen the same type in the male. The patient, who was desperately ill, had half his thyroid gland removed, and I believe that the operation saved his life, though he nearly succumbed. But the remaining half is active enough, and the mental condition is characteristic. Energy is the leading feature. He loves an argument and is full of fight. He would be in the trenches now if he could get there. He has so many irons in the fire that the fire itself is in constant danger of being extinguished.

It is evident then, that this condition of mental alertness and energy is dependent upon thyroid activity. I have noticed that the administration of thyroid extract may have a most marked influence on the disposition of children. Under its influence they sometimes become much more energetic and excited, this change being manifestly the result of increased mental activity. Conversely, does it not appear probable that the mental efforts which we are all making, and which our parents have been making for many generations before us, and which the children of successive generations are being called upon to make in ever-increasing proportion—I say, does it not follow that these efforts must make increasing demands upon the thyroid glands of the race? Under these conditions we should naturally expect the thyroid gland to increase in capacity of function as the years roll on. And I think we see the results of this increasing activity in the large proportion of so-called neurasthenia with which the practitioners of to-day are confronted. We see it also, I think, in the many highly strung, excitable children of to-day. Children who lie awake till their parents go to bed; who do sums and other lessons in their sleep, or suffer from somnambulism. Children that are always demanding excitement, and inquiring what they are going to do next. Many of these are tall for their age. They are usually thin. It is easy to believe that increased growth and thinness are the direct result of

excessive thyroid activity. It is not easy to see how calcium can at the same time be eliminated in excess. Yet this apparently occurred in the case of the overgrown boy already mentioned. But in any case it is not difficult to understand that the drain on the calcium supplies is likely to diminish the alkalinity of the saliva, which, as I have striven to show, is an all-important factor in the causation of dental caries. In support of this argument, I shall end by a reference to an observation of Professor Pickerill's. After dwelling on the remarkable freedom from dental caries shown by the Maori race in general, he states that he examined the teeth of a number of Maori school children living under European conditions, and found that 95 per cent. of them had decayed teeth. The teeth of these children decayed under conditions of civilisation and not before. The only other change that could be operative in this direction is diet, which, as I have already shown, does not adequately explain the vicissitudes of dental caries. And further, as one would naturally try to supply the children with the same kind of food as that to which they were accustomed, it is likely that diet was the least important of the changes to which these children were subjected. And the greatest change would undoubtedly be brain-work.

Finally, then, we may sum up the conclusions at which we have arrived. They are:—

(1) That dental caries can only occur when the saliva is defective in alkalinity.

(2) That defective alkalinity is the result of errors in calcium metabolism.

(3) That such errors depend not on deficient supplies of calcium, but either on defective power of assimilation or on excessive excretion.

(4) That although dental caries is prone to occur at times when there is an obvious strain upon calcium metabolism, yet uncivilised races are able to pass through these times unscathed, though we cannot do so.

(5) That brain-work is the keynote of civilisation, and as brain-work is dependent upon adequate thyroid activity, it is probable that the increased thought demanded by life under modern conditions and by modern education is responsible for increased thyroid activity.

(6) That this increased thyroid activity either causes increased elimination of calcium, or perhaps by interaction with the other ductless glands prevents the proper assimilation of calcium, and thus leads to defective alkalinity of the saliva, and so permits the occurrence of dental caries.

Odontological Section.

April 26, 1915.

Dr. A. W. W. BAKER, President of the Section, in the Chair.

Adjourned Discussion on Dr. Ewan Waller's Paper, "The Influence of the Thyroid Gland on Dental Caries."

MR. J. G. TURNER, opening the discussion by request, said that Dr. Waller concluded that dental caries could only occur where the saliva was defective in alkalinity, and that the defect in alkalinity was due to deficiency of lime salts in the saliva. Could Dr. Waller tell them in quantities by how much the saliva was deficient in lime salts? 0.5 as compared with 1 per cent. gave a 100 per cent. increase, but might yet make no practical difference. No one urging the importance of alkaline saliva had as yet shown that the percentage differences were capable of practical results. In effect they said the differences existed, therefore it was the causal agent of dental caries. To his (Mr. Turner's) mind, whatever might be the theoretical possibilities of plenteous and alkaline saliva, clinical observation did not support the idea of its supreme importance. Tartar, a deposit of lime salts from the saliva, was to be found in every mouth from childhood to old age, and in all conditions of health and sickness, including cretinism, &c. That was to say, the lime salts had been there and had been deposited actually on the carious teeth and often immediately adjacent to the caries, but their influence had been *nil*, or at least had been overborne by some other factor. This factor he believed to be germ-carbohydrate stagnation. Sticky, fine-ground cereals, starchy food well cooked till the cellulose envelope of every starch grain was broken, both well mixed with sugar, were the staple food of civilised peoples. During mastication the fine grains were forced into every crevice of the teeth and between the abutment surfaces, and were deposited on every open stagnation area, as at the necks of the teeth. The mechanical cleansing of eating could not reach them and their stickiness defied the solvent or cleansing powers of the saliva. Next they were covered by a coating of mucus and their occlusion was complete. The ptyalin of the saliva already mixed with

them turned their starch to sugar and the germs were free to make acids. Now unless saliva could dissolve or coagulate mucus, and unless this solvent action was followed up by mechanical friction to remove the sticky food deposit, it was of no more use than mouth-washes, or water to clean a duck's back. The flushing power of a flow of saliva, or of a mouth-wash, on sticky food was *nil*. The lime salts did not penetrate the occluding layer of mucus, and at best the flow of saliva was intermittent and of short duration; all they could hope from it was that it would dissolve or coagulate a certain amount of mucus and that its occluded ptyalin would turn some of the starch to soluble sugar, rendering the disintegrating sticky debris more easily removable. To the possible solvent or coagulant action of lime salts in solution Mr. Turner attributed the slight benefit observed in respect of dental caries in hard water areas, and Dr. Pearce, Tuberculosis Officer in Rochester, had told him that he noticed a marked lessening in the incidence of pyorrhœa alveolaris in hard water areas. The conditions created by the occlusion of sticky, starchy and sugary food were beyond all the powers of plenteous and alkaline saliva. In its absence caries was small in amount or absent. The Esquimau of a hundred years ago ate meat and blubber, and for three months in the year a species of berry. The few skulls (eighteen) of this age the speaker had seen were free from caries, and Mr. J. H. Mummery noted the same fact. Now they were adding molasses and fine flour to their diet and decay was becoming common. Lately he was given three totally carious teeth from one Esquimau feeding on this diet. Here there seemed to be no question of change in saliva. They still ate meat, blubber and berries, but had added molasses and flour, because these things suited their palates. The Gauchos of the plains of Argentina at one time fed only on meat and drank tea. This Mr. Turner had at first hand from two old inhabitants of Argentina. Now meat was more valuable they were getting hard biscuits served to them. His informants agreed with Mr. J. H. Mummery's statement that fifty years ago caries was unknown among them, but said that it was rare now, though it did exist. Turning to the position of caries in individual teeth, he found that when fine-ground cereals were used caries occurred commonly at the abutment points and in the minutest crown fissures—i.e., in places where fine particles alone could stagnate, and where occlusion immediately followed, while where coarse-ground cereals were used caries occurred only in such places as coarse débris could lodge—i.e., at the necks of the teeth and in large developmental pits on the crowns—and, further, was slow and less extensive. The teeth of South Sea islanders well exhibited the incidence of cervical caries dependent on coarse stagnation, and they were peoples of whose diet Pickerill, he believed, approved. On these points he had published extensive data—in a paper read before the last International Medical Congress in London and in a post-graduate lecture published in the reports of the Royal Dental Hospital of London, 1913; unfortunately, no extracts of these reports had been circulated in the dental journals. All the data he had collected went to show that stickiness and occlusion were the important points in dental caries, and that

plenteous alkaline saliva was powerless in their presence. By providing drainage—in its broadest sense—they could obviate dental caries: and if in the same mouth provision was made on one side for drainage and not on the other, the eventual result was caries, pyorrhœa and tartar on the undrained side but not on the other. This could be seen in cases of one-sided extraction of first permanent molars at about the age of 16. At the age of 45 he had found the extracted side sound, with perhaps a little tartar, but on the unextracted side had found much caries, pyorrhœa and tartar. On the extracted side the teeth had separated and twisted, the molars moving forward, the incisors, canine and bicuspids backward, till there were no, or almost no, abutment surfaces; but since at this age the roots of the teeth were formed and the bone was hard enough to resist compression, the teeth had not been merely crushed together into a smaller arch by the action of lips and cheeks as happened at an earlier age. The result was ease of mechanical cleaning—i.e., drainage. There was no question here of a better supply of saliva on the extracted side. To this point he also drew attention in the post-graduate lecture above quoted. Dr. Waller urged that caries of teeth was due to a deficiency of lime salts in the saliva and talked of lean years and periods of stress. Mr. Turner answered that the presence of tartar was proof that there was no deficiency. The lime salts were there, and if their functions were to prevent caries they always failed us. Dr. Waller would have us cast diet to the winds as a primary cause of dental caries. The speaker answered that there was very strong evidence that diet was the primary cause, and that its sticky quality over-rode all salivary differences. He might point out also, that Dr. Waller had not taken account of the properties of mucus or of the behaviour of saliva in relation to mucus—for the oral fluids were largely supplied by the mucous glands of the gums, cheeks and palate, and this mucous secretion was far the more continuous secretion.

He would now criticise Dr. Waller's paper in detail. Dr. Waller asserted that dental caries was far more prevalent and acute in early life, and claimed that only calcium deficiency could explain this difference. One might ask, incidentally, how many healthy children this allowed in the civilised world. Acuteness was at once explained by the fact that the dentine of young teeth was more organic than in older teeth. As to prevalence, the stagnation theory required that caries should at once attack the teeth, since from the moment of eruption they were exposed to the conditions favourable to its onset. Later, when the permanent teeth were in situ, drainage due to extraction might alter the incidence of caries, while greater care in cleaning and less sweet-eating were by no means negligible factors. Moreover, the expression "far more prevalent in early life" conveyed an entirely wrong impression. As a clinical fact, caries was very prevalent in early life and continued to be very prevalent through the whole of life, till either the teeth were lost or cleanliness was properly observed; wherever there was germ-carbohydrate stagnation at any time of life there would be caries. The liability to caries in absence of precaution was equal at all times of life. Dr. Waller found difficulty in the marked

difference as to liability to caries shown by the various groups of teeth. No such grouping was of any value unless it were drawn from the same subjects observed throughout life. It would be found, then, that there was some reason for separating the lower incisors from the rest of the teeth, but that all the rest might be included in one group. The lower incisors were certainly less liable to caries, he believed on account of their simpler shape, but they were far from immune. There had only to be a little crowding or hypoplastic pitting to induce plentiful decay in early life, and later cervical caries and caries of the root occurred commonly as the gum receded owing to chronic alveolar destruction and the stagnation area encroached on the roots. Dr. Waller's remark as to caries beginning on the apex (? cutting edge) of lower incisors was quite contrary to Mr. Turner's experience. So also was his statement that caries more often commenced on the coronal surfaces of teeth than between them. Probably interstitial cavities outnumbered coronal, but as coronal caries was a matter of the number of fissures and pits on the crown, there might be five or six small foci at first, and later but one cavity where all had run together, while interstitial cavities would long remain distinct. Dr. Waller found another difficulty in arrested caries, and in caries of pregnancy, lactation, ill-health, and disease. True, arrested caries was really only an extremely slow form of caries rendered so by breaking away of enamel and grinding smooth of the exposed carious dentine, or by close apposition of teeth rendering access of fluid and fresh pabulum difficult. Of the arrested caries to which Dr. Waller appeared to allude—that said to be due to recovery from ill-health—Mr. Turner had no experience, though he had looked for it for twenty-five years. Similarly, he had had no experience of caries due to disease (tuberculosis, diabetes, &c.), pregnancy, or lactation. He asserted deliberately that it was impossible to show that such persons were either more or less prone to caries than others. Moreover, caries took time to develop. Perhaps in an adult tooth twelve months might elapse after the first enamel attack before any appreciable inroad was made in the dentine, and the probability was that the decay attributed to these conditions was in full swing before they arose. He had examined the teeth of a considerable number of young cretins, and had not found them worse than other children's teeth, indeed, he found sound temporary molars persisting to the age of 16, and was thus able to demonstrate the retarded shedding and eruption of teeth due to cretinism. He could at that moment call to mind three patients with Graves' disease having good teeth as far as caries was concerned, but bad in respect of tartar and general sepsis, and he might remind Dr. Waller that there was very strong clinical proof that dental sepsis was a cause of Graves' disease. Dr. Waller asserted that in young children the temporary molars were the first to decay, and when the lower front teeth decayed he suggested it was the result of early extraction of temporary molars. In Mr. Turner's experience the molars were by no means always the first to decay, nor was extraction of molars a necessary antecedent to decay of the lower front temporary teeth. He had, however, found a history of sucking a sugar-bag or of putting softened biscuit into the mouth by rubbing

it over the closed teeth in cases of extensive caries of temporary front upper and lower teeth. Dr. Waller said it was surprising how often irregularly placed teeth remained sound; to this the speaker could only reply that they did not often remain sound. He thought premolars ought to decay infinitely more than upper incisors, but they did not. The answer was that both decayed to the utmost of their capacity, but that the premolars having cusped, and consequently fissured, coronal surfaces showed a greater incidence where individual cavities were counted. He thought the lower first permanent molars erupted before the uppers, but Mr. Turner did not think so much before as to affect the question either way, nor did he think they suffered more than the corresponding upper teeth as was usually asserted. Dr. Waller's statements as to eruption of bicuspids were, he thought, erroneous. Their eruption varied greatly, especially as it was often hastened by early extraction of septic temporary molars. Dr. Waller made much of the saliva bath for the lower incisors. This bath Dr. Waller thought was destroyed when molars were extracted. If they looked, however, at the normal pose of the head they would see that the front part of the floor of the mouth was always the lowest by a considerable margin. Moreover, the lower front teeth were prone to cervical and root decay, even in presence of tartar deposits, to quite as great an extent as other teeth. As to the suggestion that a plate preserved them, the fact was very much the contrary. Dr. Waller said caries was not always symmetrical. As a fact, it was remarkably symmetrical. His remarks on the feeding of other races were too sweeping. Who were the Mohammedans feeding on rice? So many different races and feeders were Mohammedans. Were the Fiji Islanders and New Zealanders cannibals? At most only occasionally. Further, uncivilised races had not always good teeth; 20 per cent. of carious teeth was not uncommon, and the increase in caries ran parallel with sticky, starchy and sugary food, so that the kind of food mattered considerably. Fiji Islanders' skulls, and some rice-eaters' skulls (i.e., in the main rice-eaters) from Burmah, lately presented to the Royal College of Surgeons' Museum, showed in some cases very extensive caries, and "natives" fed on European diet soon developed all the conditions of dental sepsis, including caries. Here he must ask Dr. Waller whether he had any ground for suggesting that the Maori children were fed on "native diet," or that their brains were overworked. He suggested that exactly the opposite conditions obtained.

He had criticised adversely, but he was at one with Dr. Waller in desiring to know what lay behind the food factor: and whether there was any process such as symbiosis or antagonism, or some alteration in mucous or salivary secretions, which could be controlled and so render them immune, apart from daily cleanliness. Dr. Waller was breaking new ground, and Mr. Turner felt almost apologetic in making his criticism, but Dr. Waller appeared to misapprehend many points connected with dental caries, and so found difficulty where none existed. There was, he believed, no fact of dental caries which did not fit in with the germ-carbohydrate theory. By intelligent application of the theory, prevention was possible, and as yet there was no other known means of prevention.

Dr. SIM WALLACE said that as Mr. Turner had criticised Dr. Ewan Waller's paper so much on the lines on which he himself might have criticised it, he would only refer to one point. Dr. Waller had used a peculiar theory in support of one of his arguments—namely, that Nasmyth's membrane acted as a dialysing membrane, thereby transferring the salts in the saliva to the enamel and thus hardening it. As, however, Nasmyth's membrane was soon rubbed off the enamel where it was subjected to friction, and as these situations were seldom the sites of decay, it would appear that the fact of Nasmyth's membrane not having had much opportunity of acting as a dialysing membrane conferred greater immunity to caries than the sites where Nasmyth's membrane remained longest on the enamel—that was to say, in the crevices of and between the teeth. Although he (Dr. Sim Wallace) saw no reason for believing that Nasmyth's membrane acted as a dialysing membrane, he considered that Dr. Joseph Head's theory that the enamel was hardened by the salts in the saliva was justified by the experiments which that investigator made to prove this.

Mr. J. F. COLYER said that with regard to Dr. Ewan Waller's paper, he had read it through very carefully, and he did not think that Dr. Waller had adduced much evidence in favour of the view he had promulgated. As regards the question of arrested caries, it was well known that the condition could be brought about quite easily by placing the teeth in a correct environment, and he did not think that the condition of arrested caries in any way supported Dr. Waller's views. Again, if insufficiency of the thyroid was responsible for caries, one would have expected to find a prevalence of caries in under-developed children, which was frequently traceable to thyroid trouble, but such was by no means the case. He did not think that the question of caries in animals, especially in horses, could be explained on the grounds of inadequacy of thyroid secretion. It was a well-known fact, of course, that horses developed a large amount of caries, but this was due to the lodgment of carbohydrate food, (1) between the teeth, and (2) in small canals which occasionally persisted on the occluding surfaces of the upper molars. With regard to the statement made that early extraction of the deciduous teeth was likely to lead to caries in the anterior teeth, there was not the slightest evidence in favour of that view—in fact, everything was to the contrary. In mouths where the deciduous teeth were carious and where the first permanent molars also showed caries, owing largely to stagnation of food upon them, the removal of the deciduous teeth had the effect of rendering the rest of the mouth functional, with the result that the molars were naturally cleansed in the process of mastication, and the liability to caries was lessened. The statement which Dr. Waller had made to account for the increased prevalence of caries upon the grounds of the greater strain upon the brain, was a little difficult to explain in the light of caries even as seen to occur two hundred years ago. If a series of skulls of individuals living in London about two hundred years ago were examined it would be found that

the amount of caries was very considerably less than at the present day, but he did not think that that condition could be accounted for simply on the lines of increased mental strain.

Dr. WALLER (in reply) thanked the Section for the honour they had conferred upon him by their invitation to read a paper before the Society. He was also grateful for their criticism, because criticism, even if adverse, helped one to arrive at the truth. But on certain points he should challenge his critics. Mr. Turner had denied that caries was more prevalent during youth than in maturer life. Probably Mr. Turner did not now study text-books, but if he would refer to Smale and Colyer's "Diseases and Injuries of the Teeth," second edition, he would find the statement that "Caries is more active during the period of growth of the individual than subsequently" (p. 243). With regard to the relative immunity from caries possessed by the lower front teeth as compared with the upper, Mr. Turner's argument that this depended on their shape was old, but most unconvincing. Mr. Turner also denied that caries more often commenced on the occluding surface. In answer to that, he was requested once more to refer to the same page of the same text-book, where he would find the statement that "Caries nearly always commences on the occluding surface." The statement referred to the first permanent molars. Mr. Turner objected to his statement that irregularly placed teeth (which would naturally afford the greatest facility for the retention of carbohydrate material) nevertheless frequently escaped caries. In reply, Dr. Waller could only assure him that one of the first lessons he had learned about teeth was the evil of irregularity in consequence of this increased tendency to decay, but he had many times been surprised to find that such irregular teeth often remained sound even when others were carious. If he might be pardoned for mentioning personal details, he was himself the possessor of thirty teeth nineteen of which contained fillings. But among the remaining eleven non-carious teeth were the most crowded and the only overlapping teeth in the jaw. He had looked in a great number of mouths and noticed the same thing in many of them. The presence of caries seemed to depend far more upon other factors which he had already discussed than upon mere irregularity.

Both Mr. Turner and Mr. Colyer said that the great remedy against caries was the production of free drainage by judicious extraction, scaling, and so forth, to prevent the stagnation of food. It seemed to Dr. Waller that this was tantamount to giving free access to the beneficial alkaline saliva, which was thus enabled to neutralise the destructive acids before they could do harm. In any case, the fact that caries could be arrested or prevented by this process *without change of diet* was in itself an argument that diet was not the principal factor in the causation of caries. The obvious inference, investigation of which would be profitable, was that caries was due to overcrowding of the teeth owing to inadequate development of the jaw, and that such overcrowding, by preventing adequate access of the saliva to the interstices between the teeth,

deprived the teeth of their natural protection against caries. Mr. Turner had also contended that caries was not more prevalent in ill-health than under other conditions. Probably Mr. Turner was too busy to read the *British Dental Journal*, but if he would look up the recent paper by Dr. Broderick, to which Dr. Waller had already referred, he would find that Dr. Broderick had investigated this point, with reference to tuberculosis, in a sanatorium,¹ and found that the patients were broadly divisible into two classes—namely, those with average and those with shockingly bad teeth. In the latter class he invariably found the patients that were not doing well. With regard to the first type of patient with average or even good teeth, one need not be surprised at finding many relatively trivial cases of tuberculosis in a sanatorium. Early diagnosis of tubercle was now being very much insisted upon, but, on the other hand, it had been abundantly demonstrated in the post-mortem room that practically everyone was infected with tuberculosis. There was therefore no reason to suppose that the teeth of the mild cases in the sanatorium would be vastly different from those of the mild or recovered but un-notified cases outside the sanatorium.

Mr. Turner also said that in young cretins the teeth were often sound. Dr. Waller did not wish to dispute this, though it was usually stated that in cretinism the teeth were remarkably bad. Dr. Ord, in his description of the cretin in Clifford Allbutt's "System of Medicine," said: "The lips, coarse, protruding and gaping, give a glimpse of a swollen tongue appearing between two rows of carious teeth." Dr. Waller had himself removed temporary molars from a cretin aged over 40. The essential effect of thyroid inadequacy starting early in life was retardation of development, both of teeth and bones. Hence very little calcium was needed for the skeleton. It was therefore quite conceivable that in some cases the saliva might remain adequately alkaline and so help to preserve the teeth. But the teeth were usually so late in erupting in cretinism that it was quite probable that in the "young cretins" observed by Mr. Turner the eruption of the teeth was too recent for caries to have occurred. Dr. Sim Wallace had expressed his approval of Mr. Turner's criticism, and the same answer must therefore be tendered to him. But he had also criticised the suggestion that Nasmyth's membrane could act as a dialyser. Dr. Waller was not responsible for that suggestion, which he had merely quoted from Professor Pickerill, who was able to take care of himself.

Mr. Colyer had challenged Dr. Waller as to who would get best results in treatment, Mr. Colyer by looking after drainage, or Dr. Waller by the administration of thyroid extract. But, unfortunately, the control of calcium metabolism, and hence of the alkalinity of the saliva, could not yet be achieved by so simple a process as the administration of thyroid extract. Mr. Colyer asked whether Dr. Waller would consider that caries in the teeth of the London horse was due to thyroid deficiency. In reply, Dr. Waller would ask whether it was due to faulty diet. Was the food of the horse insufficiently fibrous, too

¹ *Brit. Dental Journ.*, January 15, 1915, p. 63.

soft or pappy, too finely ground, too much cooked, or too highly saccharine? But perhaps the horse was also suffering the penalties of civilisation. At any rate, the London horse must have far more occupation and much more strain on its nervous system than the wild horse of the prairie. But on that point Dr. Waller did not wish to be taken too seriously. Equally he did not wish to be taken too seriously when he said that he wished to cast diet to the winds as the cause of dental caries. The phrase was an hyperbole. But he had intentionally used a strong expression, because so many of the dental profession could think of nothing else but diet, and he hoped to startle them into the idea that possibly there might be some other factor worthy of their consideration. There were various other points in Mr. Turner's criticism which were open to argument or even denial, but he must not trespass further upon the time of the Section. They had heard his paper or had had an opportunity of reading it, and they had heard the remarks of his critics, so he must leave judgment to them.

The Principles of Dietetics.

By J. SIM WALLACE, D.Sc., M.D., L.D.S.

SOME fifteen years ago the study of the ætiology of dental caries led me to conclude that there must be something radically wrong with dietetic habits which were current in civilised communities. Further, it became impressed upon me that the dietetic precepts emanating from the highest quarters were on the whole productive of harm rather than good, for it was by no means apparent that dental caries and other diseases resulting from bad dietetic habits were less common among the children of professors of physiology or among the children of medical men generally, than among the children of the relatively ignorant poor. In fact, in time it became evident that the teeth of the children of poor peasants, and even of those living in slums, were rather less ravaged by caries than were the teeth of those who were supposed to be enlightened in the science of dietetics. Naturally, I tried to inform myself on the subject. Among other things, I bought a book on "Food and the Principles of Dietetics," and therein found quite a mass of information on the chemistry of foods and certain physiological details associated therewith, but I found remarkably little about the principles of dietetics. Indeed, if I remember rightly, the author of this misnamed manual seemed quite to approve of the dictum that one man's meat was another man's poison, which rather than being a principle of dietetics, seemed to me to imply a negation of all principles. I have not recently tried to gain knowledge from books of this nature, being convinced that masses of scientific detail which are not directed by philosophical thinking are on the whole productive of more harm than good. It is not, therefore, my intention to treat the subject as it is treated in books on dietetics, but rather to indicate that the principles of dietetics branch naturally from the tree of knowledge and bear fruit which is or may become of great value.

I shall endeavour to state some principles of dietetics and add but a few words to each of them, as it is not possible in the short time at my disposal to do more. Nor, until the acumen of your criticisms makes me wiser, would it be advisable to attempt to do more. The first principle I would refer to is that—

The kind of food for which man is adapted is the same in all

essentials as that on which he has subsisted for countless generations. This principle is lacking in direct practical utility, firstly, because we may not know exactly the kind of food on which primitive man and his ancestors did subsist. Secondly, because we are not all agreed with regard to what are the essential elements of food; and thirdly, because man has introduced methods of preparation of foods which, though in some cases they may destroy or impair certain essential qualities, so far improve others that a reversion to primitive foods would be a retrograde step. The value of constantly bearing in mind this important principle, however, will be seen from some of the principles which follow, for without this one a certain guide for the search for others would be lacking. Here let me say a word with regard to the essential qualities of food. Generally, it is believed that the essential elements of food are that they should be capable of being transformed into nourishment for tissue building and repair, or for the production in the body of heat and energy. This may be considered the basic assumption of most physiologists. Recently it has been suggested that vitamins should be regarded as essential elements in food, though how they benefit tissue building or the production of energy has not been worked out. Similarly, bacteria and bulk, or ballast, have been regarded as essential, though how far they are so has been more or less obscured by an almost exclusive study of nutritive qualities. All that I would say here is, that such a conception of what are the essential elements of food shows a great lack of knowledge of certain points that have been evident to dentists for many years. It has been evident to all of us who regarded dental caries as a diet, or rather a dirt, disease, that an essential element in diet is generally lacking in the diet of the civilised, though it was present in the diet of primitive man. Continued study of this point showed that this essential element had nothing whatever to do with nutrition. We shall consider this subject later; in the meantime it is sufficient to note that food should not be unhygienic, in other words, that food should not induce stagnation or fermentation and putrefactive processes which the normal physiological reactions of the alimentary canal are unable to control.

The next principle to which attention may be briefly directed is that *the anatomy of the organs of digestion indicates the type of food which is most generally suitable for the animal under consideration.* This principle has long been recognised by those who have concerned themselves with palæontological zoology. It has, however, been most completely disregarded by medical experts, who have occupied

themselves with framing the pap dietary for young children which still too generally holds sway. It is now many years since the dental profession recognised that the complete temporary dentition indicated that the most suitable diet for a child was not pap, but should be of such a nature that the teeth and jaws would require to be used. Moreover, we recognised still further that the coming into position of the milk teeth indicated how the transition from the purely milk diet to the solid and varied diet suitable for children with a complete dentition should be brought about. At one time there may have been a certain amount of indefiniteness with regard to what might be considered a suitable diet for a child with a complete set of teeth. For many years a large number of the dental profession at least claimed that the food should be hard, so that the teeth might have sufficient use. Further study, however, made it evident that the use of the word "hard" was not quite *à propos*. Bones, chocolate, toffee, &c., can quite properly be designated hard, but such foods could not, in our opinion, be considered generally suitable for children. It happens that there is a peculiarity in the dentition of man, and, as far as we can see, in those of his ancestors, which makes it evident that the masticatory organs of man are not specially suited for such hard foods. A carnivorous animal, whose teeth are specially adapted for crushing bone or tearing flesh from it, does not possess the power of lateral motion of the jaws such as is more especially characteristic among the herbivora. The temporo-mandibular articulation indicates this, as does also the occlusion of the teeth. Even our nearest relations, the anthropoids, have not the power of grinding food such as man has. The canine tooth in man is not raised above its fellows so as to prevent lateral motion during mastication, and this anatomical peculiarity appears to have existed coevally with man. Here we may say parenthetically, that we must not lay too much stress on the type of food which present-day anthropoids eat. They may have been driven to live on trees by their more intelligent cousins the progenitors of man, and their food habits and dentition specialised accordingly. The food, then, which will stimulate the use of the teeth and be most thoroughly disintegrated by lateral motion during mastication is fibrous rather than hard. Fish and meat, vegetables, and many fruits (all of which we dentists are fairly well agreed are desirable from the point of view of dental hygiene), may be called fibrous, but we should be speaking loosely to call them hard. Furthermore, it is undesirable to say that hard food is suitable for children, because it is only fibrous food which can be relied on to clean the teeth, for the

juices and particles incorporated in the fibrillar meshwork are expressed during mastication, and the more fibrous part which is left last in the mouth is converted into a kind of mop which helps materially to keep the teeth and gums clean. An important confirmation of this view has recently been made by Mr. Brook Nicholls [5] in a communication dealing with the state of the teeth of the children living in the Bass Strait. Selecting the most general characteristic of a food as a basis for generalisation is useful, but it should be noted here that the separation or isolation in thought of different constituents in the food and considering them separately is liable to give rise to erroneous teaching. We must, for example, remember that "most vegetable foods, especially fruits, very generally associate cellulose, sugars, acids, and acrid principles, and it is scarcely right to consider the effects of any one of them when dissociated from the others."

A consideration of the above leads us naturally to the third principle which we have to note—namely, that the *physiology of the organs of digestion indicates to some extent the type of food suitable for man*. To begin with, however, what is the outstanding physiological process for which the first part of the alimentary canal is adapted? The incisor teeth incise or cut off portions of food and the molars crush, tear, or physically disintegrate it, while at the same time the saliva and mucus are secreted. The muscles of mastication concerned with the closing of the mouth are physiologically adapted for the exercise of enormous pressure in order to disintegrate the food, while the muscles which open the mouth are relatively weak. If the food were intended to be swallowed without undergoing physical disintegration in the mouth there would be no need for the large muscles and elaborate physiological co-ordination which at present (necessarily) exist. As far as the digestion of food is concerned, the obvious inference is that, with regard to the mouth and teeth the physical disintegration of the food is practically its one and only function, notwithstanding the fact that we may possess books written on "The Work of the Digestive Glands," in which the salivary glands are largely referred to. Do not let us be befooled into believing that the physiological *raison d'être* of the salivary glands is solely or even primarily for the sake of digesting food. The salivary glands exist and function for the purpose of facilitating the movements of mouth and food, for facilitating disintegration, for facilitating deglutition, and for helping to clean it out of the mouth as rapidly as possible after the disintegration of the food is sufficiently advanced. We have heard a great deal about the digestion of starch by ptyalin.

and although the amount is very trivial it was well dinned into us that the function of the ptyalin was to digest starch (for nutritional purposes). That a copious flow of saliva is poured out when sugar is taken into the mouth did not seem to suggest the idea that the function of the saliva was to clear substances out of the mouth which might be or might become harmful to the teeth and mucous membranes during or after mastication. So again, we were taught that acid vegetable foods gave rise to a copious flow of alkaline saliva rich in ptyalin, but physiologists were silent with regard to the "why" of the ptyalin in this case. That the flow of alkaline saliva was to neutralise the acid was indicated, but that the saliva similarly became alkaline on eating meat was not mentioned, and the rationale of such alkalinity was not indicated. So, too, we were led by physiologists to believe that the reason why ptyalin did not exist in the saliva of an infant till it had reached about the sixth month was that up to this age the child should not get starch in its dietary. No doubt an infant should not get starch in its dietary before this age, but why a ferment from the pancreas capable of digesting starch before this age existed was not told to us. The fact that the appearance of ptyalin in the saliva synchronises with the cutting of the teeth and that the saliva exists chiefly for the physiological cleanliness of the mouth and for the protection and preservation of the teeth was apparently never dreamt of. Although, then, the physiological reactions of the saliva to food show us clearly that they are adapted chiefly for facilitating disintegration of food and deglutition and for getting rid of certain substances commonly associated with foods which are or might become injurious to the mouth and teeth, it would be a mistake to argue that whatever stimulates the salivary glands should necessarily be regarded as specially desirable food. Rather should we surmise that in foods which stimulate a great flow of saliva there may be an element of danger if for any reason the glands were not capable of responding adequately to the stimulus or if the food were of such a nature as to hamper the saliva in its attempt to get it out of the mouth. There is, then, an element of danger in food which contains sugar in greater proportion than it exists in vegetable foods as presented in Nature, and the physiology of the glands of oral hygiene indicates that sweets, bread and jam, bread and marmalade, and the like, should not be eaten at the end of or between meals.

I have not referred to mucus. Its function is largely that of lubrication, but it, too, is also instrumental in maintaining hygiene of the mouth, and when thoroughly incorporated with the food during mastica-

tion it no doubt facilitates the action of the gastric juice when the food reaches the stomach. Possibly this explains to some extent why, as Dr. Boyd has ably shown, digestion is so very much better when the food requires mastication than when it is given in the form of pap.

Here it may be noted that the various principles of dietetics cannot be altogether isolated from one another, or perhaps we should say that various principles serve to reinforce each other, so it need hardly surprise us that our fourth principle has been adumbrated in what has already been said. This fourth principle is most important not only because of its practical application but also because it has hitherto been completely overlooked by physiologists. I refer to the principle that *the food should be of such a nature or the meal so arranged that the mouth and teeth will be left physiologically clean at the end of the meal.* When once this principle is stated no one can deny it, for it is simply absurd to say that the mouth should be left in an unhygienic state after meals. In my earlier writings I have so frequently insisted on the importance of this principle that I shall not even trouble you with a recapitulation of the facts and arguments which commend its importance. The wholesale destruction by caries of the teeth in civilised communities, together with the equally wholesale destruction of the gums and alveolar processes resulting from oral mal-hygiene, should have been sufficient to call attention to the importance of this principle, and except among those physiologists and medical men who consider that the mouth and teeth are outside their domain, the principle is now fairly recognised amongst the educated. Since the teeth of animals and primitive man are kept in a hygienic state by their foods, it is a simple guide to say that food in its natural condition will, if taken at the end of a meal, leave the mouth clean. For various reasons I have drawn attention to the value of uncooked fruit for this purpose, and need only add here that unless we are to revert to primitive foods and eschew all the benefits which accrue from the preparation and cooking of foods, we must above all other principles insist on this one. Moreover, recognition that food should be of such a nature that the mouth and teeth should be left in a hygienic state leads by extension to the idea that the whole alimentary canal should be left in a similar hygienic state, and this suggests a still wider field for investigation. I have read that the alimentary canal of children nowadays is little more than a series of pathological specimens, and although this may be an exaggerated way of putting it, there is too much truth in the remark; and so long as the craze for nutrition and calorie values is considered to the exclusion of the hygienic value of food, what truth there is in that observation will remain.

“ Science knows no geographical boundaries ” ; it only recognises the limits of its own departments. Being under less than no obligation, however, to follow decadent scientific methods, we may be permitted to transgress the boundaries of our specialty and deal briefly with a few principles which have little or nothing to do with the mouth and teeth. They have, however, to do with the hygiene of the alimentary canal. As over-eating is a common cause of alimentary mal-hygiene, I shall deal with a few principles associated therewith. The first I shall mention is well known but seldom brought into force, as it should be among the civilised. It is simply that “ *hunger is the best sauce.* ” Throughout the whole course of evolution our progenitors must often have been hungry, sometimes very, very hungry. If hunger had incapacitated them for food when it was found, there would have been little chance for them surviving ; fortunately, it had quite the contrary effect. Pavlov [6] has shown by approved experimental methods that digestion goes on best when associated with hunger, and in a general way we all know that it does so. Nevertheless, I have practically never seen an emaciated child with an unhygienic digestive tract treated by keeping food away from it, and the unhygienic state of their alimentary canal is never given a chance of becoming hygienic. The administration of tonics and digestive stimulants generally increases appetite, tending to over-eating, and thereby to derange a weakened digestion and thus inducing further mal-hygiene, while the administration of anti-septics is an admission of the existence of an unhygienic state without attempting to remove the cause. Combined, these drugs indicate the nature of the scientific preparations which exhibit our ignorance of the principles of dietetics.

This leads us to state another principle—namely, *tempting the palate habitually is contrary to the dictates of evolution.* As the importance of economy in nutrition has, throughout all ages, only been transcended by the importance of getting enough, we should recognise that a flagging appetite is a signal that amply sufficient has been consumed. We may be assured that, as in past ages, unlimited supplies of food are seldom available, as much as possible will be eaten of any food which contains the elements for supplying immediate needs and a reasonable reserve for the immediate future without special temptation. One might have thought that a scientist who recognised that hunger was the best sauce might also have recognised this principle. Nevertheless, we read in Pavlov’s work on the digestive glands : “ The customs of the chief meal of the day also correspond with our physiological results.

After this or that *hors d'œuvre*, perhaps also with a liqueur of brandy (especially customary in Russia), both of which are designed to awaken the appetite, the repast proper begins, and, in the majority of cases, with something hot, consisting mostly of meat broths (*bouillon*, different soups, and so on). After this comes the really nourishing food—meat of different kinds served in various ways, or, in the case of poorer people, stews made with vegetables, and therefore rich in carbohydrate material. This sequence of foods, from the standpoint of physiology, is quite rational. Meat broth, as we have already seen, is an important chemical excitant of gastric secretion. An attempt is therefore made in two ways to secure a free secretion of gastric juice to act on the chief food; first, in the excitement of the appetite juice by the *hors d'œuvre*, and secondly, in the promotion of the flow by the action of the meat broth." It would appear from this quotation, and other similar statements, that no effort, either in the way of chemical or psychic excitant, has to be spared to secure the activity of what seems to be presumed an otherwise insufficient organ for carrying on the work of digestion. Pavlov's [6] ideas are interesting and instructive; unfortunately, however, they are not principles of dietetics; rather are they principles of gluttony. Much that is written about the arrangement of the meal from the glutton's point of view may be interpreted quite differently and more correctly. The meat or vegetable broth which so frequently commences a meal may have little or nothing to do with stimulating digestion, it may have chiefly to do with economy. A proportion of the available nutriment in yesterday's joint might be lost if the remains were not boiled and the resulting "stock" used on the following day. The words "stock" and "*pot au feu*" both suggest this interpretation. Pavlov follows what I have already quoted with the sentence, "The usual termination of the repast is also, from the physiological standpoint, easy to be understood. The chief meal is generally ended with something sweet, and everybody knows that sweets are pleasant. The meaning of this is easy to guess. The repast, begun with pleasure, consequent on the pressing need for food, must also, notwithstanding the stilling of hunger, be terminated with an agreeable sensation." Let us consider what is meant by "something sweet." Jam rolls, bread and honey, chocolate, &c., may all be recognised as something sweet or sweets, but yet, as it appears to me, his meaning in saying that the meal should be terminated by something sweet, yet which does not burden the work of the alimentary canal at this time, is not at all clear. It is quite

obscure unless (which may be his meaning) we assume that sweet things do not burden digestion at this stage. But this would be quite erroneous. He refers also to acids, and attempts to show how acids one way and another assist digestion. He never appears to have dreamed that acids in foods were almost wholly concerned with dental hygiene. In fact, the whole of his arguments indicate a conviction in the insufficiency of man's organs of digestion, except when they are excited, whetted, and tempted by all sorts of psychic and chemical stimulants, and the philosophy of the correct termination of the meal is overlooked.

That the gustatory secretions should be stimulated at the end of a meal not by sweets but by food of a natural, hygienic or detergent nature is evident to all who admit the principle that the mouth should be left physiologically clean. Now what does so most effectually? Fresh fruit certainly does: not only the dilute sugars, but the acids, the aromatic and even the acrid substance in the fruit do so most effectually. Fruit contains but little nutriment and its real value is hygienic and not nutritive, much less is it a digestive tonic. Foods in the form of sweets, jam rolls, bread and marmalade, chocolate, &c., are to be condemned absolutely as meal terminals, as their effect is the opposite of fruit, which leaves the mouth in a hygienic state. Read in the light of my theory Pavlov's experiments are, I believe, most instructive. Read in the darkness of the old craze for nutrition they are misleading, dangerous, and contradictory.

The amount of food eaten should not be regulated by principles of gluttony. It should be regulated indirectly by recognising the principles of dietetics. That the palate is a useful guide is not to be denied and when it is given a chance to act it acts usefully. If the palate is given the opportunity of discovering and appreciating the quality and quantity of the food which passes it, as when the food requires to be disintegrated and retained in the mouth for a time, then it will be a useful guide. If, on the other hand, food is given in the form of pap and is slipped past the palate without mastication, as it so often is in the feeding of young children, and of geese which are being stuffed for *pâté de foie gras*, then the palate is but an imperfect guide both as regards quality and quantity. It may be said that we want to have a guide for future meals and want to know beforehand the quality and quantity required for individuals under diverse circumstances. If the principles of dietetics indicated are recognised this is easy, as the amount required for to-morrow is under similar circumstances the same

as that which was required for yesterday. To avoid too little being provided, provision should be made that a little of a wholesome but not specially tempting kind shall always be left over, and arrangements made for utilising it on the following day, or for feeding animals or otherwise. You will no doubt say, "But this is just what everyone does," and how everyone does regulate the amount provided daily. Of course it is. My thesis here is that common-sense and custom are generally better guides than standard dietaries, scientifically based on an erroneous assumption. Nevertheless, let me say that instinct, custom and common-sense are not invariably to be relied upon. A new environment may come into existence relatively suddenly and the safeguards of necessary limitations existing in the aboriginal environment may disappear. Thus, for example, a relatively unlimited supply of carbohydrate foods may now be easily procured; in addition to this a gratuitous advocacy in influential quarters of the things most to be guarded against may usurp the place of natural limitations, as, for example, the advocacy of highly nutritious foods, the value of sugar as an economical foodstuff or as a proteid sparer, the advocacy of jam and marmalade instead of butter on bread, or the extolling of cocoa and chocolate to the prejudice of cheaper and more hygienic foods. It is for reasons such as these that we stand in need of something more than instinct and custom. We need the principles of dietetics and a touch of common-sense to be able to co-ordinate and make practical use of them.

With regard to a balanced dietary little need be said; the principles which regulate this, too, are similar to those which regulate the amount. Like the amount, it is very variable, and if the organism (when restraints such as Nature presents are imposed) is not fit to seek for and procure the balanced dietary required no artificial endeavours will be likely to avail in the long run. The possibility of getting a fair choice and variety is of course necessary. Variety, however, should not simply be with regard to proteid, carbohydrate, fat, &c.; it should include also *uncooked* food. Fresh fruit, vegetables and gustatory stimulants without any regard to calories are necessary. Throughout the whole course of evolution uncooked food was consumed, and we certainly have not as yet sufficient evidence to make us disregard the probability that certain essential qualities are not destroyed by cooking. Possibly the craze for having everything made easily digestible and sterilised before it enters the mouth accounts for much dyspepsia, and may explain why uncooked milk, though full of germs, is such a

useful drug in restoring what is sometimes necessary for healthy digestion. I indicated above that restrictions on eating certain foods should be imposed to compensate for the lost restraints which Nature has imposed. If restraints in the nature of taxation for revenue on consuming alcohol, tea and coffee are conducive to more good than evil, then surely the taxation of sugar, sweets, chocolate and cocoa might for similar reasons be imposed, for, as Sir William Osler has maintained, decayed teeth are responsible for greater harm to the community than alcohol. Surely the proportion of misery which alcohol causes is not greater than the amount of dental troubles brought on by the eating of sweets. Indeed, Dr. Wheatley's [8] statistics with regard to sweet eating and caries clearly show this to be the case.

The next principle we may refer to is that *diet and activity are reciprocally co-ordinated*. This is generally recognised, and so we need only refer to the modern doctrine which runs counter to it—namely, that the worry and strain of modern civilisation is responsible for much of the prevalent dyspepsia and other ills to which human flesh is heir. The doctrine is generally applied with regard to the well-to-do and more often than not to Society ladies whose so-called work and worry resolves itself into making numerous calls and indulging in the light chatter which is characteristic of drawing-room conversation. We fail to see the great strain involved in this; but that those who indulge in it are frequently the subjects of dyspeptic derangements admits of little doubt. The explanation of this fact is probably that the work necessarily entailed on their ancestors in gathering, preparing and cooking food, in cleaning and looking after the house and helping in the work of tending and bringing up children, has been got rid of by the employment of servants, and the idle time thereby gained is misused on introspection, on gossip about the physiology of the alimentary canal and other internal parts, and about the nutritive value of the latest advertised foods containing concentrated nervous restoratives required for the upkeep of the highly strung machine which has been "run down" by the heavy strain and serious worry which gossip and the failings of servants involve. It has struck me as suggestive that the wives of drunkards and wastrels seem generally to have remarkably good health and digestion. The wives of such men have much to worry about, but their worry is about things external to themselves. They are necessarily very much concerned with their environment, for the effects of drunkenness loom large on their horizon. Fortunately,

they have no time to worry about their own physiology, nor leisure for morbid introspection. To cut a long story short, there are two forms of worry—one is external, natural and beneficent. This latter has been the common lot of humanity throughout the whole of man's struggle for existence. It is the mainspring of work and of most healthy activities. The other form of worry, which is harmful, is introspective, and dependent for its existence on idleness and morbid states of mind and body. This last form of worry is injurious, and its proper antidote is work, not prescribed gratuitously as a medicine but as a necessity. Civilisation and division of labour largely dispense with the necessity of the actual work of procuring and preparing food before meals; still, some form of activity should precede or alternate with meals. Present-day substitutes, such as physical exercises, breathing exercises, skin friction and ablutions regularly performed before breakfast, have considerable justification from the point of view of evolution. The point in which they fail is that they are done without interest, and without seeing that the activity subserves some useful end, except the rather morbid one of the conscious pursuit of health. There are many activities which would no doubt lead to as satisfactory results if indulged in before breakfast, and the fact that domestic servants, notwithstanding the shocking state of their teeth and the illnesses resulting therefrom, are as a rule relatively healthy, makes us surmise that this type of activity might well be imitated by women and even men in affluent circumstances. It would also, to a certain extent, get rid of the fanciful idea of the "worry and strain" involved in keeping domestic servants.

The next principle we may refer to is that *economy in diet is beneficial physically and morally*. A long sermon might be preached on this subject; it is not, however, my intention to do so, firstly, because it would be more instructive to hear how it is done from the housewife with a family, let us say of five children and a husband with thirty shillings a week. Secondly, because it involves other considerations besides calorie or hygienic values. Thirdly, because the poor housewife in general does exceedingly well with the money at her disposal compared with those who usually give advice to this class. She does not require to be told that bread, oatmeal and potatoes are economical compared with many other foods often belauded in medical journals, nor that cheap cuts of meat or cheap cheeses are of as high nutritive values as the more expensive kinds. All that I would say with regard to this principle is that to judge the value of a food by

laboratory tests regarding its calorie value has been, and is, most misleading. A food such as uncooked fruit may have a high hygienic value. This may at times be wanted, and what is not wanted, even though it has calorie value, is valueless when it is not wanted. Moreover, the estimation of the cheapness of food by the amount of calories procurable from a given sum has led to the wholesale distribution of advice which is worse than useless. Because an amount of sugar representing a large number of calories can be bought for a relatively small amount, it has been argued that sugar is an important and valuable food *for the poor*. Since, however, force of circumstances compels the poor to obtain the great bulk of their nourishment from bread or cereals and potatoes, and inasmuch as these contain relatively a superfluity of carbohydrates, every penny spent on sugar by the poor is from the point of view of nutrition simply wasted. But this is not all. The sugar is often taken in a particularly obnoxious form, either as sweets between meals or as jam spread upon bread. Not only, then, is the money spent on the sugar wasted, but the fruit which is made into jam has most of its virtues destroyed as far as oral hygiene is concerned, and this at the considerable expense which is entailed in reducing it from the hygienic and palatable form in which Nature provides it to the unhygienic form in which we see it prepared to destroy the teeth.

Another principle which should be considered by those who issue dietetic precepts is that *functional activity is generally necessary for the most perfect development of an organ*. A conspicuous illustration of this may be seen in the muscular development of the blacksmith's arm. This principle applies also with regard to the development of the salivary gland and probably also to other glands for the hygiene of the alimentary canal. Similarly, without any doubt, the muscular development of the organs of digestion is affected by the physical nature of the food. This is notably so in the case of the muscles of mastication ; moreover, the development of the muscles of mastication has a distinct influence on the development of the jaws, and this again has its influence on the arrangement of the teeth. The anatomical perfection, then, of the first part at least of the digestive apparatus of man is quite appreciably affected by the physical nature of the food consumed in childhood.

I might refer to other principles which indicate, for example, that eating between meals should be avoided ; that the concentration of the different elements composing foods (especially sugar) should not exceed

the concentration habitually found in such foods as man must have subsisted upon throughout his evolution ; that three meals a day are to be preferred to any greater number, &c. But as these and other principles dealt with in this paper have been gone into in much greater detail in my books there is no need for further elaboration at present.

I should, in conclusion, however, like to refer to some reasons for bringing forward this communication. One general reason is to indicate that principles of dietetics may be formulated. Nevertheless, we need not be too sure that those whose business it is to deal with diseased states will formulate them, because if there are any principles with regard to dietetics among the diseased, they would by no means necessarily be the principles which obtain in health—indeed, we may perhaps truly say that one diseased man's food is another diseased man's poison. Similarly the principles of dietetics or the value of foods cannot be established by those who set up laboratory tests and standards as criteria. Nor can they be deduced from the most elaborate statistics having reference to calorie values. The accumulation of statistics on nutritive values, however accurate, gives no more guide to hygienic values than do hygienic values give guide to nutritive values, and the use of such statistics as a basis for generalising only prevents us from doing what science ought to do—namely, to methodise knowledge and reduce it to principles. Further we must be practical. Principles are merely of use as mental formulæ for the sake of doing something for human welfare. The pursuit of science for science's sake is, as a rule, merely an excuse for inability to discover principles which are of human utility. My special reason for bringing this communication forward is because I feel confident that all members of this Section have in their own practice seen the awful havoc which has been wrought in the mouth and more especially the teeth of the people through the misguided teachings of those who have in the past expounded the principles of dietetics to the medical profession and so indirectly to the public. I feel sure that each in his own way will try to bring home to the teachers of medical students the urgency of perhaps the most important, though as yet unappreciated, principle that I have brought forward this evening—namely, that the meals should be of such a nature that they will leave the mouth physiologically clean. I know it is difficult to gain the ear of those who stand on high pedestals. I know that it may be galling for those who teach and think they are leaders of thought to be themselves taught and to have their own thoughts led. Yet the dental profession is undoubtedly at present able to direct the

teachings of scientists, as regards diet; indeed, it has been said that "in the last few years the diet of children has been entirely reconsidered. Much of the traditional feeding has been rejected, and in its place a carefully thought-out dietary, based on scientific principles, has been established," and that, moreover, "it is to the dental profession that in the main we owe these advances." Similarly, it has been admitted that "We have now quite outgrown the notion that dietaries can be scientifically constructed by a mere estimation of calorific values." Referring, however, to the oddly unfair attitude taken up by many with regard to some of the most signal triumphs of preventive medicine, a reviewer of a book said, "Dental disease is a striking instance of this attitude. Without demanding any absurdly stringent regulations of one's daily life, without asking any expenditure of public money, dental disease can be almost entirely prevented. But how little stock is taken of this striking triumph of our art. . . . It is useless waiting for the heads of the profession to take it up; the general practitioner can do much to bring about the change."

But these observations have been made by individual medical men. I have not yet seen such admissions made by accredited teachers of medical students. Nevertheless, until the distinguished professors of physiology appreciate and teach the principles of oral and alimentary hygiene, the teaching of which could prevent more suffering than any hitherto discovered physiological principle, it will take us a long time to effect the result for which both principles and professors should exist. It must surely stimulate all of us to think that we are in the position of those pioneers of science who, having brought forward and elucidated new thoughts, stimulate fruitful investigation. Our case is amply sufficient, not only to justify but to compel careful and accredited workers in physiology and physiological chemistry to turn away from blind-alley researches to the scientific investigations of numerous details connected with the subject of this communication in the new light that promises the most fruitful returns. It may be that in the immediate future we shall be treated as common dentists whose directions are to be regarded with disdain. Or it may be that the roads that are leading to fruitful results will be claimed to have been opened up by those who have been directed by us. That need not disturb us at present. What we want first is to see that the teeth of each succeeding generation are less and less ravaged by decay. We want to be able to note that the ugliness resulting from ill-developed jaws, and ruined and misplaced teeth, which blight every second face we look upon at present, will

gradually give place to beauty of form and fitness in function of this conspicuous and important part of the body. We want to be able to show that pyorrhœa, with all its odours and disastrous results, is being controlled and made rare, and finally we want to see dyspepsia, resulting from continuous dietetic error, more or less completely banished.

I am convinced that all this can and will be done by all intelligent and moderately careful people, *provided the true principles of dietetics are elucidated by those in influential places.*

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DISCUSSION.

Dr. HARRY CAMPBELL agreed with most of Dr. Sim Wallace's remarks. Laboratory experiments had done little to teach them dietetic principles likely to be of practical use, and it was just such principles of which the medical man stood most in need. He thought they might obtain many useful practical hints by studying the changes which man's diet had undergone in the course of his evolution; and especially by comparing the diet of primitive peoples with that of modern civilised communities, notably the inhabitants of this country. It was important to bear in mind that, before man learnt to cook his food, all his starchy food (the supply of which was, moreover, limited) was subjected to laborious trituration in the mouth. The study of the condition of the teeth of the anthropoid apes was of great practical interest in this connexion; the extent to which they were worn down—even the milk teeth—showed to what laborious use they were put. An anthropoid skull duly studied from this point of view was worth a whole course of lectures on medicine.¹ The introduction of cooking, by means of which the cellulose was disintegrated and the starch capsules ruptured, lessened the need for such laborious mastication. Primitive man, long after he had learnt to cook his food, continued, however, to masticate much at least of his vegetable food thoroughly, as shown by the wearing down of his teeth, and this was even true of the inhabitants of many parts of

¹ See the Anthropoid Skulls in the Museum of the Royal College of Surgeons.

Europe. The effect of such thorough mastication was not only to exercise the jaws and teeth, but to produce a thorough insalivation of the starch. Dr. Sim Wallace doubted whether the saliva had any appreciable effect in facilitating starch digestion. He (Dr. Campbell), on the contrary, was in the habit of teaching that a considerable degree of starch digestion could be effected in the mouth by thorough mastication. How striking was the contrast between pre-cooking man, who subjected his limited intake of starch to sustained trituration, and the modern Briton, who sucked his food rather than masticated it, and who overwhelmed his stomach with starchy food consumed in a liquid, pappy, pultaceous, or spongy form. The study of man's past, and the study of the mammalia in general, showed that it was the starchy food which demanded thorough mastication. All the frugivorous and herbivorous mammalia were laborious masticators; all the carnivora, bolters. Hence the golden rule that starchy food should be consumed (above all in the case of children) not in the form of soft bread, buns, puddings, porridge, and the like, but in a form (such as crusty bread) which compelled mastication. When this great truth was assimilated by the medical profession, and its application enforced, an enormous amount of disease of the teeth, nose, throat, stomach, and bowel would be prevented.

Dr. BECKETT-OVERY said he had much pleasure in congratulating Dr. Sim Wallace on the interest and importance of his paper. This was especially the case because the speaker had come to very much the same conclusions from an entirely different standpoint—namely, that of the general practitioner dealing with the ordinary run of diseases. He was absolutely certain that to people living more or less inactive and sedentary lives too much starch was most pernicious. Dr. Harry Campbell had referred to the probability that the anthropoid apes, who were the precursors of man, became carnivorous. In connexion with this it must be remembered that both they and carnivorous animals ate their flesh raw and with the blood, thus retaining the very necessary constituents of the blood, which, in the speaker's opinion, were absolutely essential. The interesting point was that these salts could be made up to a large extent by means of the leaf vegetables, if cooked conservatively, or raw as salad, and the speaker suggested that from Dr. Sim Wallace's point of view salads would achieve the same purpose as fruit, which, in the speaker's opinion, did not always "agree" with meat. Raw fruit was usually better taken apart from meat. The speaker cordially agreed with Dr. Sim Wallace in deploring the fact that so many of the leaders of the profession did not appreciate the importance of this question of diet. There was a possibility of reform in this direction being forced on the profession from outside.

Mr. T. P. BEDDOES said that the aim of dietetics was to devise suitable diets, to supply sufficient nourishment, to allow of the greatest amount of work, and of the maintenance or recovery of the best health at the lowest cost. The teaching of physiology was a good guide, to a limited extent, but must be supplemented by practical experience, and it did not come within the scope

of the physiologist to discuss details, apart from main principles, of oral hygiene, or the hygiene of any special orifice. Some physiologists were not members of the medical profession. None could be described as heads of the profession of whom an unnamed reviewer of an unnamed book in an unnamed journal was quoted as saying: "It is useless waiting for the heads of the profession to take up this striking triumph of our art." There was an advantage in dietetic investigations on animals. The cost, weight, and chemical composition of their less varied food were more easily estimated: the weight of animals when alive and when dead could be determined. The profit or loss on some investigations was a financial matter. What one regretted in the advice of Dr. Sim Wallace was that he had little to suggest except the taking of detergent, fibrous food, and fruit at the end of a meal. Fruit was a somewhat vague expression to those who could not procure it; and, in the case of those who could, it might conduce to the evil against which he warned—i.e., over-eating; for it was not after a light lunch, but after a heavy dinner, that fruit, in the form of dessert, was usually taken. It was undesirable to praise or decry unduly the advantage of estimation of calorie value of foods. Calories were of actuarial use in estimating the chemical composition and cost of food, but as a basis of diet they required to be checked by practical experience. In the feeding of horses their worth had been proved by obtaining the greatest amount of work together with the best health at the lowest cost. Again, to take milk as an example. This was a necessity for infants whose mothers could not suckle them or could not suckle at the usual time, and in certain countries, where the suckling was prolonged to the second or third year. Excluding these cases, laboratory estimation of calorie value showed that milk was an extravagant diet.¹ A further consideration showed that improper preparation of food might destroy its nutritive value. The value of milk might be impaired by over-heating, and rickets, scurvy-rickets, and scurvy might thus be set up in infants. In recent investigations on beri-beri, as important a disease in the Tropics as typhoid fever was in cold climates, calorie value was not mentioned.² In one instance the introduction, through lay influence, of fruit into the diet led to bad results, which continued until use was made of the experience gained from laboratory investigations on the feeding animals.

Dr. SIM WALLACE (in reply) said: In reference to the question put by the President, he had no intention of indicating that the septic condition of domestic servants' mouths did not tend to induce ill-health, but rather that in spite of this domestic servants as a class were relatively healthy because of the amount of work they had to do. With regard to Dr. Harry Campbell's observations, he would only say that when he said they were not all agreed with regard to the exact nature of the food of the ancestors of man he referred generally to what was known. He (Dr. Sim Wallace) agreed that Dr. Harry

¹ Alan Murray, "Economy of Food," 1911.

² W. P. Chamberlain, *Journ. Amer. Med. Assoc.*, April 10, 1915, p. 1215.

Campbell had shown in his writings on the evolution of man's diet what must have been the nature of the food which the ancestors of man consumed. Unfortunately there appeared to be many who were not acquainted with his writings. He was glad to note that he was in agreement with Dr. Harry Campbell with regard to the general harmfulness of milk and sweets. With regard to the observations of Dr. Beckett-Overy, he was very gratified to learn that he, too, had independently arrived at conclusions similar to those contained in the paper he had just read. Concerning the possibility of the incompatibility of meat and raw fruit, he could only say that for about the last ten years he had been advocating fruit as a termination to the meal of his patients, and had not observed that those who followed this advice suffered from indigestion on that account, although in the case of adults there were many cases in which the digestion had already been deranged, and fruit of any kind could not be indulged in either with or without meat. With regard to Mr. Beddoes's criticism, it would appear that he (Dr. Sim Wallace) had been flogging a dead horse. Perhaps the work containing the tenets with which he had found fault most was Dr. Hutchison's book on "Food, and the Principles of Dietetics," but he did not know that Dr. Hutchison's book was generally considered out of date. Commenting upon Mr. Beddoes's statement that physiologists had rather abandoned the estimation of calories in favour of experiment on animals, he would only say that the ultimate test was whether human beings benefited from the knowledge gained by such experiments. And from his own experience, confirmed by what Dr. Harry Campbell and Dr. Beckett-Overy had just said, it would appear that even where the so-called best advice was obtainable, the results on the upbringing of children were at least pathetic, and the statement that the alimentary canal was too often a series of pathological specimens seemed to be confirmed. Notwithstanding Mr. Beddoes's remarks he (Dr. Sim Wallace) was still of the belief that no published book by any living physiologist had ever even mentioned the subject of oral hygiene.

Odontological Section.

May 31, 1915.

Mr. J. H. BADCOCK, Vice-President of the Section, in the Chair.

A Note on Extraction.

By J. G. TURNER, F.R.C.S., L.D.S.

WHEN our Secretary asked me to fill a gap to-night it occurred to me that a short discussion on extraction might not prove uninteresting. The pain of extraction is dependent on three factors: The sensitiveness of the tooth itself, including the pain of rupture of the nerve; the tenderness of the surrounding parts—that is, the acuteness of any inflammation there may be; and the extent of injury to surrounding parts done in extracting. The pain of rupturing the nerve is unavoidable, and, moreover, is surprisingly small. To minimise the two latter sources of pain we must proceed with the object of inflicting the least amount of injury possible. In extracting by levering the tooth from side to side the bone is sprung open or perhaps broken, and considerable pain is produced by crushing the nerves running in it. In extracting by “going wide” and including gum, bone, and tooth, great pain is inflicted, and most hideous and slow-healing, painful wounds are left, while very often the apex of the tooth is left in. A little consideration will show the reason for this. We do not know in either jaw the length of the root we are dealing with. The mandible is cased with thick layers of compact bone, increasing in thickness towards the base. The alveolus of the maxilla widens rapidly upwards, especially on the palate side, where it also has a not inconsiderable casing of hard bone.

The only way I know to avoid these difficulties is to extract by displacement. If we can push the blades of the forceps along the

sides of the root, between it and the bone, the tooth will be dislodged. Forceps for this purpose must have much finer blades than the root forceps sold at the depots. Indeed, it is a wonder to me how roots are ever extracted with so-called root forceps; their blades are so wide that they cannot possibly pass into the tooth-pocket, they are at once stopped by the surrounding bone. Forceps for operating by displacement must have narrow, straight blades with sharp, slightly everted terminal edges, but may be left fairly thick in transverse section to give strength. In operating, the points of the blades must be applied to the sides of the tooth or root, without gripping, and still without gripping they must be pushed along the socket till the tooth is loosened, when it is gripped and taken out. This operation leaves an uninjured gum and makes a minimal displacement of bone. Difficulty is found in keeping in the axis of the tooth. If much force is being employed the first thing to suspect is that such force is being used in a wrong direction—that the blades are being pushed into the surrounding bone. The right axis must be sought for by drying the tooth or root and examining its direction. The roots that give most trouble are slender roots embedded in thick bone, but if their direction is properly gauged they will be found easy of extraction. It may be necessary to find the direction by trial, desisting when much force is being used, on the assumption that the blades are being driven into bone in a wrong direction. Forcible pushing up, also, usually means that unconsciously the handles are gripped, in which case the points of the blade are often being driven into the root. An exostosed root may be suspected where there has been chronic inflammation, and here the blades must be kept wider apart.

This method is obviously not fitted for such teeth as a first upper permanent molar with widely divergent roots, but an apparently immovable molar, upper or lower, may be reduced to a simple extraction by displacement by use of the splitting forceps, and, if needed, a burr on the engine, when the three or two roots may be dealt with separately.

In preparing for this operation care must be taken to clean the periodontal sulcus, since any germs lying there will be carried up into the socket by the forceps blades. In operating by displacement I find I cause far less pain to the patient; I leave a wound which is nothing more than the empty socket, which causes little pain, and heals well; I break off but few apices, and of those so broken I am able to get out the larger number, since they are often to be found loose at the

end of the socket, and can be hooked down with a probe or hoe-ended excavator, and I succeed with slender roots where the broad "root forceps" are hopeless failures.

I do not wish to propose this method to the exclusion of molar forceps. Indeed, I think that where a good grip can be taken of a molar by the molar forceps, they are the most suitable instruments. In the case of upper molars, especially, I can see no reason for attempting the extraction of sound-necked teeth with root forceps, especially with such as are in general use. The blades will not travel along the sockets, nor will they take a firm, extensive grip of the neck. Their success depends on brute force and entails extensive injury to bone.

Extraction by displacement with narrow-bladed forceps demands special care with "gas" anæsthesia, since at the moment the tooth is loosened the blades have no hold on it, and if it jumps out they are not wide enough to contain it.

DISCUSSION.

Mr. GEORGE THOMSON said that it seemed to him a long time since this subject had engaged the attention of this Section. The paper referred to the separation of the roots of upper molar teeth to facilitate their removal; an easier method was that of dividing with a fissure burr the palatal root, so that the buccal roots might be removed together first and the palatal root separately. Referring to the displacement of teeth, without damage to the gum or alveolar border, the speaker had on more than one occasion been questioned in legal cases as to the seriousness of injury done to the adjacent tissues in the removal of teeth. On one occasion the judge, while listening to the explanation of an operator who stated that his method was that of carefully insinuating the forceps between the gum and alveolar border without damage to the tissues, suggested that his method seemed to be that of "gentle persuasion." The speaker had replied to the judge that he had often found it necessary when one side of the tooth was broken down below the gum level to grasp both gum and alveolar border externally. He would like Mr. Turner to express an opinion as to whether he would consider this to be justifiable.

Mr. WILLIAM HERN agreed with Mr. Turner in his criticism as to the frequently improper shape and size of the blades of forceps for root extraction. Such blades were usually too thick and bulky on the convex side and this made it impossible to grasp with them a root broken within a bony socket. The groove of the blade, too, was commonly too large to pass within the socket as it should easily do. The Middlesex Hospital possessed several root forceps made by the late Mr. Everard, probably with the late Sir John Tomes as architect, which were the most efficient instruments for the removal of roots

he had seen. He agreed with Mr. Turner in his plea for extraction by displacement as he termed it. He had on occasion unscrewed and used successfully a single blade of a pair of Everard's bayonet-shaped upper root forceps for the removal of a root of an upper molar broken far into the socket. The elevator, known as Mr. Charles Rogers's, which was really the shape of a single blade of forceps, set at an angle of about 45° with its handle, was a most useful agent for the extraction of a root of a lower bicuspid when broken low down in its socket; a root could be displaced by this instrument with very little damage to the surrounding socket.

Mr. W. RUSHTON said that the value of Mr. Turner's interesting remarks would have been enhanced if he had brought for their inspection the instruments he described. His own forceps were modelled on those of Everard and, from what he gathered, they were similar in type to those suggested by Mr. Turner. Though it was important to use the best type of instrument, yet in difficult cases, such as crooked, twisted, or exostosed roots, the most important factor in success was experience, which guided them in their choice of instrument and taught them discretion in its use.

Mr. DOUBLEDAY thought that Mr. Turner's remarks about the displacement of teeth were so important that they should be emphasised. It was his (Mr. Doubleday's) practice to teach that in extraction three steps were necessary: (1) The correct application of the forceps to the root of the tooth; (2) the most important part of the operation, the dislocation, or displacement of the tooth from its attachments, by which was meant the dilatation of the alveolus or space and the rupture of the fibres of the periodontal membrane; and (3) the extraction of the tooth from its socket, which might be performed with the fingers or a conveying forceps if the other steps had been correctly carried out. Considerable force might be necessary on occasion, but it should be force directed to the dislocation of the tooth from its attachments and not to its extraction. The term "extraction" was a misleading one which might well be banished from their vocabulary, and he believed that the displacement of the teeth which Mr. Turner had impressed on them that evening was a point of the very greatest importance.

Mr. TURNER, in reply to Mr. Thomson, said he never had found it necessary to grasp both gum and alveolus in extracting teeth or roots. He condemned it as a general practice, though he could understand some operators finding it useful on occasion. One upper forceps he used was modelled on an original forceps of Everard's. He would be glad to show his forceps on a future occasion. He thanked Mr. Doubleday for his expression of appreciation.

Recent Studies in the Calcification of the Teeth.

By F. J. BENNETT, M.R.C.S., L.D.S.

IN English-speaking countries at any rate, the name of George Rainey is well known to all who have studied the formation of the lime salts in the teeth and bones. He it was who established a new point of departure by his experiments in causing the lime salts to deposit in various solutions of gum and albumen and by comparing these results with the appearances to be found in the shells of crustaceans and in the teeth. The chief point he sought to establish was that a colloidal solution was an essential feature in producing a modification of crystalline form in the lime salts, at least as regards the carbonate of lime. He described the formation of these into spherites and calculi of peculiar pattern and also their mode of disintegration.

Rainey pointed out that the usual form of crystallisation of carbonate of lime was altered from the flat rhomboidal plates to a radiate or spheroidal form, and he mentions that barium, strontium, and calcium carbonates, but not magnesium carbonate, are thus modified. Mineralogists divide the carbonates into two groups—first, the calcite series, with flat rhomboidal plates; and second, the aragonite series, with a radiate or star-like pattern, to which group barium, strontium, and sometimes calcium belong. In fact, calcium carbonate is dimorphous, and exists in two different crystalline forms. Rainey mentions that magnesium carbonate does not form a spheroid, and we see the reason for this is that it belongs to the sub-group calcite, not to the aragonite group, like barium and strontium carbonates. It is highly probable that Rainey's calculi are varieties of the aragonite series of minerals. I pointed out more than ten years ago that there was no experimental evidence in his treatise to show that these spherites could be formed artificially, where the proportion of phosphate of lime to carbonate was as high as is found in the teeth and bones. I do not question the fact that spheroidal bodies are found in developing teeth, as was pointed out by Salter and Mr. Mummery.

There is shown on the screen a drawing of Rainey's calculi. The peculiar radiate pattern is well known to mineralogists as an example of twining of crystals, the individual crystals being set at a slight angle to one another. Besides the radial markings seen in the figure, one has

to notice also a number of concentric ones. There are also to be seen the union of two or three of the spherites. For comparison is exhibited a specimen of malachite or carbonate of copper. It is seen to be a mass of spherites identical as to pattern with Rainey's spherites. The radiate and concentric markings are precisely similar; note also the round or bossy exterior. Now there is not the slightest evidence to show that these spherites of malachite are formed in a colloidal medium, and yet it was the influence of the colloid upon the crystalline form which was the principle upon which Rainey, and Ord and his school, founded all their deductions. It is fortunate that we can throw some light on the matter, and in this way: It is possible to produce malachite artificially, and though it is rather difficult yet in the process it is very easy to produce the spherites. Here is a small glass trough, at the bottom of which is a piece of chalk, a soft form of limestone. This is covered by a very weak solution of nitrate of copper in distilled water; an ordinary glass slide stands obliquely in the solution. After some weeks the slide is removed, then placed in sodium carbonate solution, and afterwards washed and mounted for microscopical examination. You may examine the specimen in this way, or from the lantern slides which I will show. You will note there are isolated spherites, and some united in pairs and triplets.

The advantage of this method is its simplicity; you have merely pure water and two chemically reacting substances. The medium is almost transparent, and it is not colloidal. In order to make a closer resemblance chloride of copper and chalk were used to produce the carbonate of copper, the copper chloride being analogous to the calcium chloride used in making calcospherites. The copper carbonate thus produced formed smaller spherites, which had a tendency to unite in groups and break up into a granular powder, much resembling the appearance of the disintegrating spherites seen in the forming layer of the shells figured by Rainey and Mr. Howard Mummery. Precipitated chalk was sometimes used in these experiments as being quite free from silica and other impurities. The reaction in all these experiments was acid at the commencement, and gradually became neutral to litmus paper.

It was during this stage of my experiments that I became acquainted with Mr. Howard Mummery's paper describing the presence of calcospherites in considerable quantity in the forming enamel; his drawings leave not the smallest doubt in my mind as to the spherites forming an important part in the process of calcification of enamel and, possibly,

in dentine also. The very high percentage of phosphate in enamel, together with the presence of spherites in such quantity, seemed to make it improbable that the spherites in the enamel were formed of carbonate of lime. This suggested further experiments to me, and having already succeeded in producing carbonate of copper spherites in aqueous solution, a similar method was adopted with the view to producing spherites of phosphate of lime. Powdered tribasic calcium phosphate was made to react with copper nitrate in distilled water. Spherites were not at first so apparent, but a web-like radiate mass of crystalline form was produced, the radii joining up with those in contact with them to form a uniform fine network. These in time grew larger

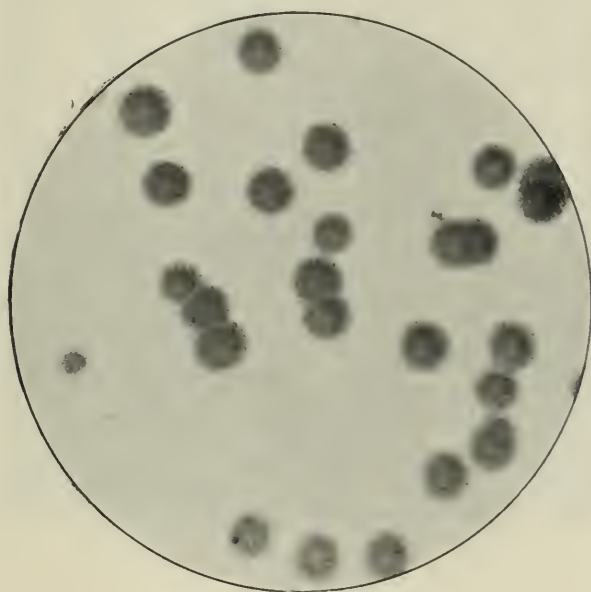


FIG. 1.

Carbonate of copper spherites formed by the action of copper nitrate solution on common chalk.

and stouter, and became completely detached from their neighbours, and, finally, in other examples, the radiate pattern formed itself into a close compact spherite. There appeared a gradual development from the fine radiate network to the closely compacted spherite. There is a considerable resemblance in the appearance of the early stage of this radiate pattern of phosphate of copper to the figures of the deposit of phosphate of lime in urine called stellar phosphate, and to the crystals of acid calcium phosphate as chemically prepared. To the naked eye a precipitate of a phosphate often falls down in smooth layers, at first uniform in appearance, but as they proceed they begin to show circular

openings or spaces, in the centre of which is found, detached, the stellar crystalline spheroid; it falls out of the layer by a continual breaking up in this manner, and the membrane-like layer becomes of a ragged, irregular pattern with small hanging tags, these in their turn forming themselves in spherites until the precipitate is entirely used up in this manner. Deposits on the glass slide and on the side of a glass beaker show this also. I have seen specimens both of copper phosphate and of calcium phosphate showing this mode of formation very clearly. From this description it is evident that the spherite itself is only a part of the process.

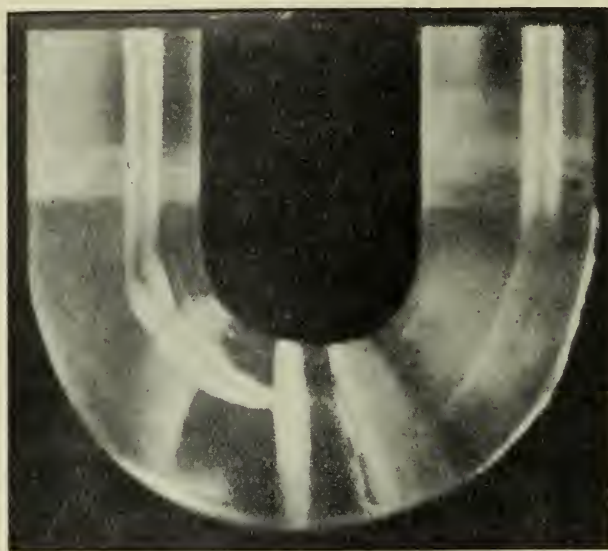


FIG. 2.

A U-shaped tube, into the lower half of which a gelatine plug is melted. When this is set, the one arm of the tube is filled with an ammoniacal solution of calcium chloride, the opposite with a solution of common sodium phosphate. These meet in the substance of the plug, forming the white bands of insoluble phosphate of lime separated by translucent bands of soluble chlorides of ammonium and sodium.

With the information thus obtained from the production of copper spherites, it may be asked if the analogy is sufficient to warrant a belief that phosphate of lime spherites can also be produced in aqueous solution. I have some specimens now under observation in which a radiate pattern has been reached, perhaps corresponding to the second stage of the copper phosphate formation seen under the microscope, but it has not yet reached the closely compacted final stage of spherite formation. Possibly I may have to alter the experiment, but

I feel sure a complete spherite of phosphate of lime in aqueous solution can be produced.

Those who have read Rainey's treatise at all carefully must have been struck with the stress he lays on the density of the solutions he causes to combine. For instance, in the experiment of the mixture of gum arabic with carbonate of potassium and water, they must together produce a solution having a specific gravity of 1460, and the solution of gum and water, which is to be poured over it, must have a specific gravity of 1084. It is a curious fact that similar attention is given to the density of the solution of nitrate of copper in the formation of artificial malachite. It therefore seems to me probable that the

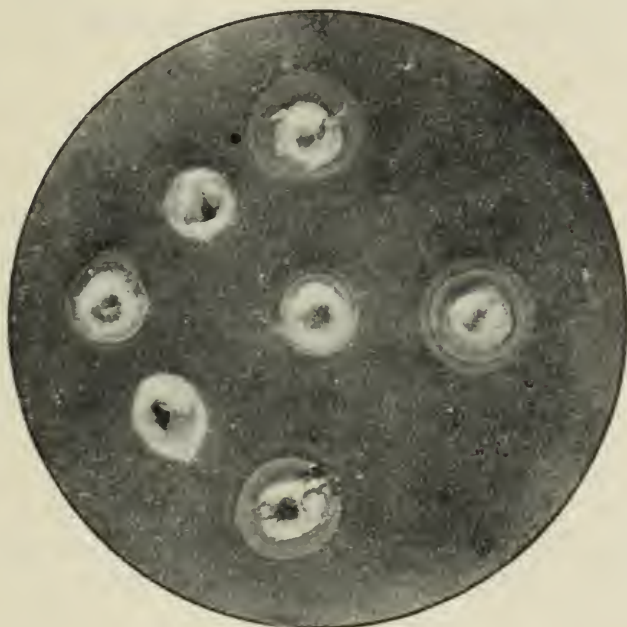


FIG. 3.

A plate of agar jelly, into which common sodium phosphate has been melted. The seven white patches are small holes cut in the agar, into which calcium chloride is placed drop by drop. The alternate rings of light and dark are due to the same causes as in fig. 2.

colloid or albumen in Rainey's solution acted by giving the necessary density.

I have succeeded in preparing some radiate forms of calcium phosphate which give a fair approximation to the appearances of the copper phosphate spherites, and this suggests that one can obtain spherites of phosphate of lime when distilled water is used, though their habit and appearance differ from the carbonate spherites.

I consider that the spherites in the enamel are formed of acid

calcium phosphate, and that their breaking up into a fine powder is due partly to the phosphate becoming neutral in reaction.

There is one point of importance in dealing with chemical reactions which take place in semi-solid substances.* Let us consider first what takes place when two chemical substances combine in aqueous solution—say, sulphate of copper and carbonate of soda: at once a uniform blue precipitate is formed of carbonate of copper and sodium sulphate. If we make the two substances combine in a gelatine plug, previously melted into the lower third of a U-shaped glass-tube, a solution of the same copper sulphate, filling one arm of the tube, and carbonate of soda solution the opposite arm, the two salts no longer combine in the uniform manner as in a simple solution in water, but a series of deposits of various colours are seen in the substance of the gelatine plug,

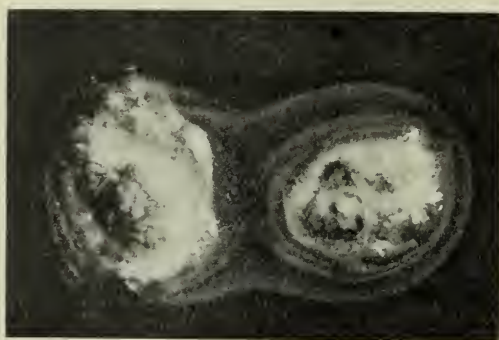


FIG. 4.

Similar to fig. 3. The inner bands, as they spread out, join with neighbouring ones, forming a new series of bands.

showing that the chemical substances have combined in various proportions to form many intermediate substances. If in a similar manner we cause, in the one arm of the tube, an ammoniacal solution of calcium chloride and in the opposite arm a solution of common sodium phosphate to pass into the gelatine plug, the result is not a uniform wall of deposit of phosphate of lime, but a series of bands of opaque white deposit, alternating with clear translucent bands. The explanation of this is that the two substances which are formed in the decomposition separate out in the plug, the white bands of insoluble phosphate of lime alternating with the translucent bands, soluble ammonium chloride (sometimes also sodium chloride with it); this may be proved by cutting out the translucent area of the plug and testing for chloride with nitrate of silver, when a white precipitate of silver chloride results.

We may produce a like result by taking an ordinary Petri dish, and melting into it gelatine, to which has been added a little phosphate of soda. After the gelatine has set, small holes are cut away in the gelatine halfway down to the bottom of the glass and an ammoniacal solution of calcium chloride instilled, drop by drop, into the small holes in the gelatine. After a few hours alternate rings of opacity and translucency appear in the gelatine in a similar manner to those in the U-tube. If two or more of the holes are made, the rings which result will in time cut each other, so that a series of separate circles are found enclosed in a larger one which encircles the two, and so on.

To a limited extent this action is found to be taking place where spherites are forming by the mere action of crystallisation, because at the moment of solidifying water is thrown out and is held between the crystals.

DISCUSSION.

Mr. J. HOWARD MUMMERY said that having been recently engaged in a study of the phenomena of calcification in dentine and enamel he was particularly interested in Mr. Bennett's communication, which he had kindly given him an opportunity of looking through before the meeting. While all were acquainted with the classical researches of Rainey, the parallel work of Professor Harting, of Utrecht, was perhaps not quite so well known. This work was independently undertaken, in fact, Harting's first publication, "On a Microscopic Study of Precipitates and their Metamorphoses," was published as early as 1840. To both these workers he considered was due the credit of original research, but he could not but think that the work of Harting was marked by more scientific method, and a more perfect knowledge of physiological processes. Rainey threw ridicule on the work of Professor Kölliker on bone and dentine, denying the existence of tubes in the dentine, and as he was apparently more or less an opponent of the cell theory, he failed to see the cells in the lacunæ of bone. Rainey would seem to have been more of a chemist than a physiologist, while Harting showed an intimate knowledge of both subjects. His lucid explanation of his different experiments was a model of scientific method. Mr. Bennett's production of spherites of copper carbonate was very interesting if these were really produced in a non-colloidal solution, although the conditions must be exceptional and perhaps capable of explanation, for it was known to be a rule that in solutions of a crystalloid such bodies did not appear. Professor Leduc, speaking of crystalloids and colloids, said: "There is no sharp line between the two groups, the passage is gradual, and it is impossible to say where one group ends and another begins. Many substances appear at one time in the crystalloid state and at another time in the colloidal state," and he speaks of these expressions as "denoting different

phases of the same substance." Mr. Mummery was as much surprised as Mr. Bennett would have been when he found large calcospherites in the laminae of growing enamel, as from a repetition of Harting's experiments in albumen he had been convinced of the fact that a large preponderance of phosphate over carbonate of calcium prevented the formation of calcospherites. Knowing the large excess of phosphates in formed enamel, one found these bodies very difficult to account for in this situation; but the large spherites were there, and they had to do what they could to explain their presence. The only explanation which he could see, and he only ventured on this as a hypothesis, was that given by Professor Sims Woodhead with regard to bone, when he said: "Newly formed bones, or new bone tissue of any kind, almost invariably have a larger proportion of carbonate of lime than fully formed bones—the active cells setting free a larger proportion of carbonic acid." Mr. Mummery, therefore, thought it was possible to consider that the spherites were deposited in a medium containing carbonate of lime in excess of the phosphate, the later addition of phosphates from the blood causing the disintegration of the spherites as the addition of phosphates does in artificial experiments. Mr. Bennett suggested another explanation, and any suggestion was welcome which tended to clear up this very obscure problem, but they had no direct evidence that this acid phosphate existed in young enamel any more than they had of the excess of carbonate. He had been much interested in the communication and hoped that Mr. Bennett would perhaps forgive him for expressing his satisfaction that he (Mr. Bennett) was a convert to the view that the globular form *is* the arrangement of the lime salts in the teeth.

Mr. BENNETT, in reply to the remarks made by Mr. Mummery, thanked him for his friendly criticism.

Odontological Section.

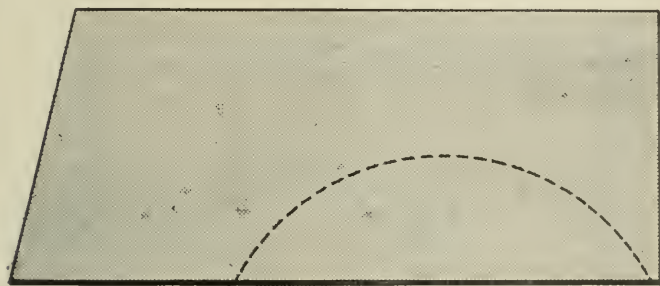
June 28, 1915.

Mr. J. H. BADCOCK, Vice-President of the Section, in the Chair.

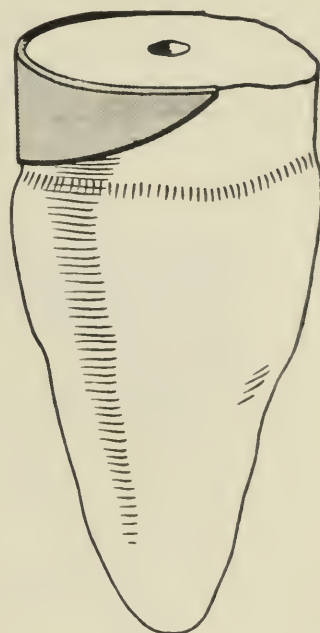
A Method of Crowning Incisor Teeth.

By GEORGE THOMSON, L.D.S.

MR. THOMSON said he used 22-carat gold, No. 4. The first step was to cut out a semicircular piece and fit that to the root. The piece was

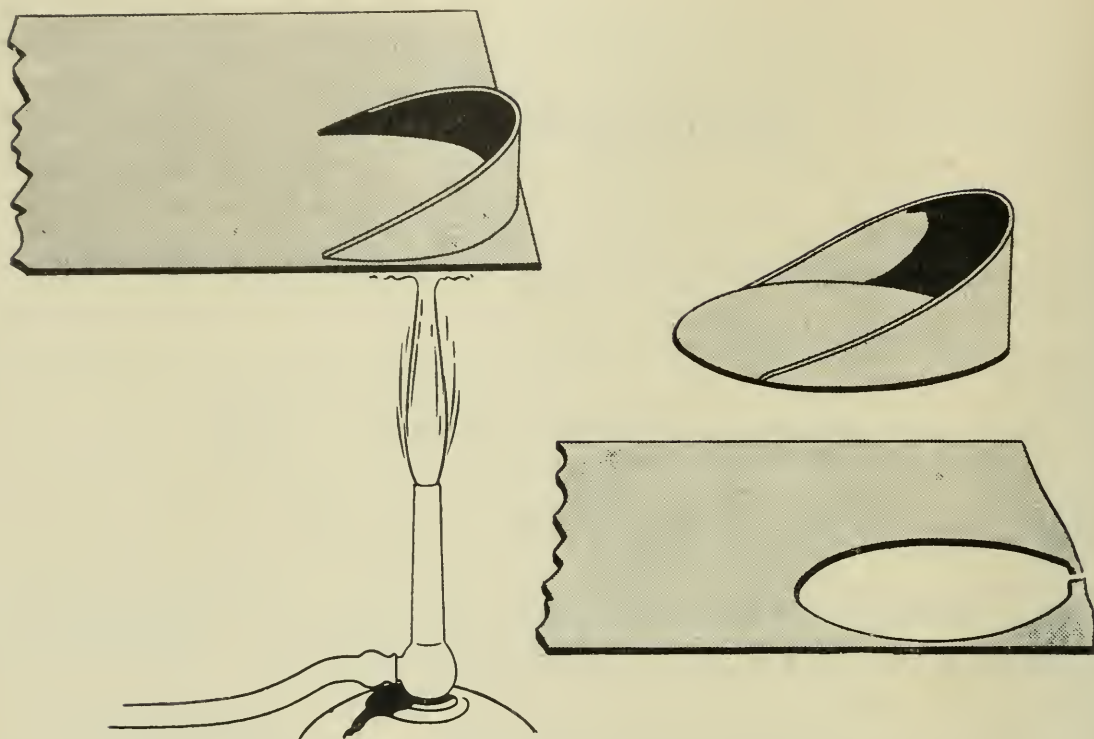


Shape to be cut out of gold.

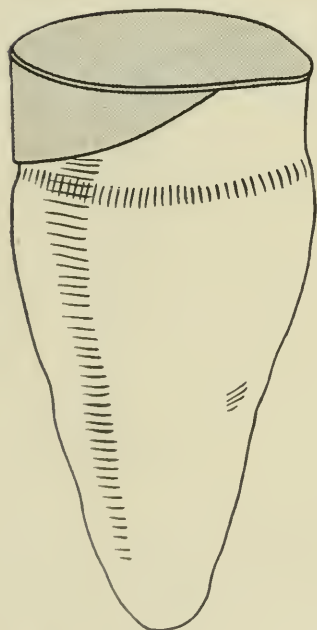


Bent round root.

straight-edged, and easily soldered over a flame; it was then cut out and closely adapted to the root. He next found the position for the pin, for which a hole was punched in the diaphragm. This was fitted to the root, and a tapering pin was forced through a hole of diameter smaller than that of the pin. He was generally fortunate in being able to draw them off together, and he then soldered them over the flame at once. He had not seen anything so simple described in the text-books.



Soldering collar to plate and cutting out diaphragm.

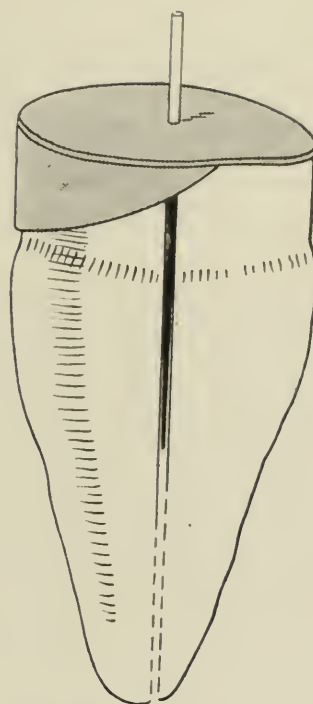
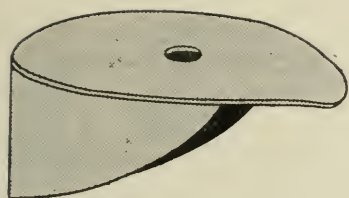


Diaphragm fitted to root.

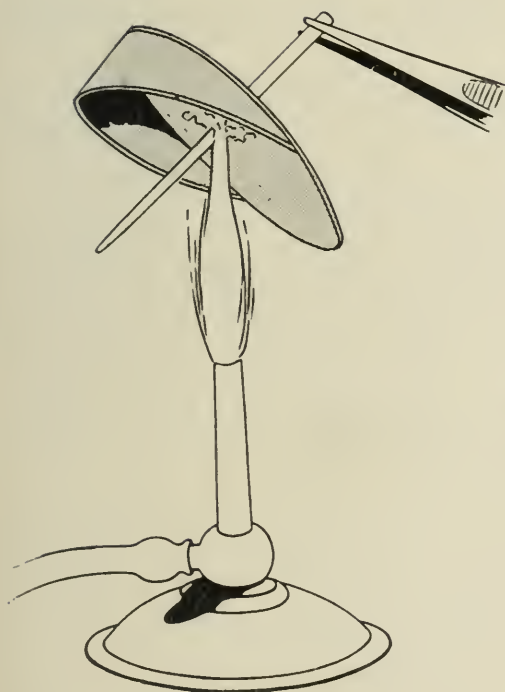


Position for pin ascertained.

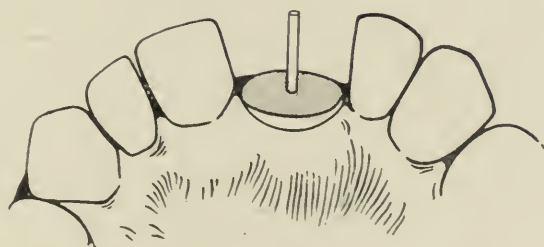
A Method of Crowning Incisor Teeth.



Hole punched in diaphragm and pin fitted.



Soldering pin.



Diaphragm fitted to root for impression to be taken.

A Method of Crowning Incisor Teeth.

The Discovery of *Entamæba buccalis* in *Pyorrhœa Pus*,
and Treatment by Emetin Hydrochloride.

By ERNEST STURRIDGE, L.D.S., D.D.S.

THE striking coincidence of the simultaneous discovery of *Entamæba buccalis* in *pyorrhœa pus* by two independent investigators living in different parts of the world has brought this subject prominently before the notice of the dental profession.

Dr. Chiavaro, of Rome, states that "entamœba is found in the pus of all cases of *pyorrhœa alveolaris*," but he is of the opinion that it "has no pathogenic action."

Dr. Barrett, of the University of Pennsylvania (supported by a number of other investigators), has advanced the theory that in view of the constant presence of the protozoa in *pyorrhœa*, and of the fact that emetin destroys the amœba and brings about an apparent cure of the disease, that *Entamæba buccalis* is "the immediate important factor in an overwhelming number of *pyorrhœa* cases."

Which of these two diametrically opposed conclusions is correct remains to be proved.

In view of the importance of the claims made by these investigators, I undertook some research on the subject, and during the past nine months have examined a great number of specimens of *pyorrhœa pus*. At first I had little success in detecting amœbæ, but after some experience and with the assistance of experts at one of the pathological laboratories, I found that the protozoa were present in nearly every case of *pyorrhœa* in which pus was visible. The number of specimens examined, of which proper statistics were kept, was fifty. In thirty-nine of these, active amœbæ were found; the remaining eleven gave negative results or were doubtful.

The method of examination consisted in transferring the specimen of pus or scrapings from *pyorrhœa* pockets to a drop of warmed normal saline solution on a slide, which was kept at body temperature over a warm water bath; the coverslip was adjusted, and within a few minutes of procuring the specimen it was placed on a warmed stage of the microscope and examined. When seen in the active state the organism is a cell of gelatinous appearance, which changes its form by projecting one or two pseudopods which extend in thick digitate

processes from the main body of the organism; it is very motile, and appears to ingest blood cells and bacteria. The nucleus is invisible in the unstained specimens. Material was obtained from patients in every stage of pyorrhœa—from advanced chronic cases, from acute early cases, from cases in which no visible pus was present, and from cases which had been previously treated and were apparently doing well. In all these amœbæ were found to be present in the proportion recorded—39 out of 50, or, say, 78 per cent.

Emetin was tried in the treatment of ten cases in which active amœbæ had been found, and the effect carefully noted. Emetin hydrochloride, put up in ampoules of $\frac{1}{2}$ gr. in 1 c.c. solution, was injected with a hypodermic syringe into the pockets. The teeth were cleansed of all foreign matter in the usual way before and during the treatment. The action of emetin in the treatment of these cases took the following course: The first and second injections seemed to have little or no effect, or else to produce a slight irritation of the gums. After the third injection pus discharge seemed to diminish and there was obvious improvement; after each subsequent injection this improvement was maintained. I did not give more than five injections in any case, as I did not consider the improvement sufficient to warrant further treatment, and there was no indication that a rapid cure was being effected. Re-examination of the contents of the pockets was also carried out after emetin had been applied three or four times, and on every occasion this was done no amœbæ were detected, but the usual number of bacteria was present.

The therapeutic value of emetin in the treatment of pyorrhœa is confined to its action on mouth protozoa, which it seems to destroy effectively. We cannot afford to ignore the discovery of the constant presence of amœbæ in pyorrhœa pus, especially at the present time, when so little is known of the real influence of this organism. But the value of emetin treatment, which is so strongly advocated in America, cannot yet be correctly estimated, on account of the short time which has elapsed since it was first introduced—only about nine months ago. It was gratifying to me to find on re-examination of many cases in which amœbæ had been found that after treatment by ionization alone, with zinc ions, the protozoa were not present. All the cases which had been experimentally treated with emetin were completed by this method.

The weak point in the emetin treatment of pyorrhœa consists in its having no effect on pathogenic micro-organisms, and unless it can be shown that the amœba is the only organism responsible for the breaking

down of the tissues emetin is not likely to do all that has been claimed for it. Nevertheless, I have prescribed tincture of ipecacuanha on several occasions (a few drops to be used with a brush once a day) in cases in which amœbæ have been found in the mouth, with apparently very beneficial result.

It has been suggested to me by Professor Kirk, of the University of Pennsylvania, that emetin might be used to better advantage by the electrolytic method. Being an alkaloid of an order similar to cocaine, its action in combination with the current would be that of penetrating the tissues to some depth. I have been trying it in this way, but am unable yet to say if it exercises any really beneficial effect.

The short time allotted me to-night does not permit of my further enlarging on the subject, but I trust the investigations I have been enabled to make will be of some interest, and I shall be glad to hear if others have tried emetin in the treatment of pyorrhœa, and with what results.

DISCUSSION.

Dr. PENFOLD said that during the last two or three weeks he had been examining a number of cases of pyorrhœa for the organism *Entamæba buccalis*, at the Royal Dental Hospital; he had been associated with Dr. Cropper in the work. Dr. Cropper had been studying amœbæ for the past eighteen months. They had had results similar to those obtained by Mr. Sturridge. Out of twelve cases examined, the organism was definitely found in eight. Their technique, however, had been somewhat different from that employed by Mr. Sturridge. Dr. Cropper and he had used Ross's jelly method—3 per cent. agar on a glass slide with $\frac{1}{2}$ per cent. salt. If a stronger salt solution were employed, they found they did not get a successful result. If they simply took material from the pocket in the ordinary way, the results were negative; the best way to take samples was to use small capillary glass tubes, bent at the end and drawn out to a fairly sharp point. These were dragged along the floor of the pockets. The material so obtained was blown on to glass coverslips, which were then inverted over agar jelly. The preparations were immediately examined. There was no difficulty in finding the amœba in many cases. The vital question was, Of what importance was the amœba in the production of pyorrhœa? Could these organisms be regarded as an ætiological factor? His colleague and he had made very few trials in the normal mouth by this method. Two, however, were so tested, and in neither did they find the amœba. In nearly all their pyorrhœa cases they got spirochætes, but not in either of these two normal mouths. With regard to treatment by emetin, the results seemed to have been contradictory, and the probability was that this substance was not the

absolute cure which it had been claimed to be. It appeared, from the literature, that in many cases the amœba could be removed easily by the use of emetin without affecting the pockets. Other factors apparently tended to maintain the disease, and sufficient evidence that the amœba was the causative organism had not yet been produced. It was difficult to distinguish these amœbæ definitely from leucocytes and pus cells. He found the amœba showed a fairly clear ectoplasm, and a definitely granular endoplasm with food granules. The type of amœboid movement in the case of the amœba was different from that of the leucocytes; the mode of progression of the leucocytes was more of a slow, dragging nature.

Dr. CROPPER said that two months ago Dr. Penfold brought to his notice the fact that he was carrying out researches on pyorrhœa, and he was glad to do what he could to help him. As Dr. Penfold had said, he (the speaker), had had eighteen months' experience in looking at amœbæ, and it would suffice if he said he had seen them along with him. Dr. Penfold had pointed out the difficulty of detecting the amœbæ and distinguishing them from leucocytes, and that was really an important point, because there was no absolutely characteristic difference or criterion between the two. With regard to the possible causal importance of the amœba in pyorrhœa, one suggestion which might be offered was, that the presence of amœbæ in the pockets did not necessarily mean that they were the cause of the condition, because they might have gone there in search of food, or to avoid the general knocking about in the mouth cavity during mastication. There were all kinds of organisms and protozoa found in pyorrhœa; even flagellate forms were seen. He was speaking that day to a colleague, Mr. Drew, who did some experiments on amœbæ, and found that there was a positive chemiotaxis between dead or living bacteria and amœbæ; therefore, if an amœba had an opportunity, it would naturally make for the place where bacteria were abundant. Possibly that was an alternative explanation of the occurrence of amœbæ in cases of pyorrhœa.

Mr. STURRIDGE, in reply, said he had been much interested in the remarks of Dr. Penfold and Dr. Cropper; but it was a peculiar fact that the organism was found in so many cases of pyorrhœa pus. His own personal view was that it had not any pathogenic action or any real influence on pyorrhœa. The mouth undoubtedly was suitable for the deposition of the amœba, for it certainly flourished there. The use of emetin seemed to exercise a beneficial effect, though not to the degree which one might expect: certainly not more than ordinary antiseptics, when the removal of all foreign matter was successfully accomplished.

The Effects of Cleft Palate Operations on the Dental Arch.

By H. BLAKEWAY, M.S.

It is convenient to deal separately with the operations which aim at directly attacking the dental arch, and with those whose influence upon the arch is indirect.

(I) Of the first class, the only operation that I am well acquainted with is Brophy's.

The most suitable cases for this operation are cases of complete clefts of both hard and soft palates, associated with hare-lip, the gap in the lip communicating with that in the palate through a more or less widely cleft alveolar process. In such cases the dental arch is already deformed, not only by being cleft, but by the premaxillary bones being displaced forwards. The displacement in bad cases of double hare-lip and cleft palate is very obvious, the premaxillæ forming a very marked protrusion beneath the nose, connected by a stalk with the anterior part of the nasal septum; in cases of single hare-lip and cleft palate, both premaxillæ remain attached to the maxilla of one side, but are separated by the cleft from the opposite maxilla (fig. 2). In spite of this attachment they are still pushed forwards by the overgrowth of the septum nasi, but being anchored on one side are more or less tilted sideways, so that the nose is displaced away from the side of the hare-lip. Brophy prefers to operate on such children at an age younger than 3 months. I have seen the results in twelve cases, and it seems to me particularly appropriate to discuss the effects of the operation upon the dental arch, because my experience has been that the effects are almost confined to the dental arch, any results upon the palate itself being usually negligible. It will be remembered that Brophy claims that the operation closes the cleft in the hard palate. I have not seen such a result; the cleft in the palate has not even been much narrowed, in spite of all the squeezing of the jaws that was justifiable; the only part of the cleft to be closed has been that in the alveolar arch, and this was not due to inexpert operating (though the operation is a complicated one requiring careful study), for two of the twelve cases were operated upon by Dr. Brophy himself.

It cannot be doubted that the closure and completion of the alveolar arch is an important result, even though the palate still remains to be

operated upon, and the question has to be decided, is it worth while or justifiable to submit an infant to an operation of the severity of Brophy's in order to bring about this result? I have decided for myself that it is not. I have never performed the operation upon a living child, and I have no present intention of doing so. The dental arch can be closed in much simpler ways, with much less danger to the child.

As regards the manner in which the shape of the arch is restored in Brophy's operation, if no more than moderate force is used in pressing the bones together, bending and not fracture occurs, for the bones, though well ossified, are soft and yielding at this early age. I have seen fracture of one alveolar process occur, but it was due to the use of too much force in the attempt to approximate the palatal processes. Some published accounts speak of necrosis of the jaw following the operation; none of the cases that I have seen had it; probably it is a theoretical objection to the operation, and theoretically it is reasonable enough, for there is always a certain amount of sepsis under the lead plates through which are passed the wires which hold the jaws together, and infection of the bone may easily occur in the course of the six weeks during which the wires are in place. The constant pressure of these plates upon the outer sides of the alveolar processes might be supposed to interfere with the growth of these latter. I have not enough evidence upon this point, but some of my casts showing the results of the operation suggest an atrophy of the jaws.

The difficulty of passing wire sutures from side to side through the alveolar arch without causing injury to the contained teeth is an evident objection to Brophy's operation. Frequently the needle is felt to strike a tooth, and its path has to be altered to avoid it. In at least two cases I have seen teeth exposed at the time of operation, and sometimes teeth are shed during the few weeks following. The final effect upon the temporary teeth is not likely to be good. In three cases which I saw respectively at the ages of 2 years 9 months, 17 months, and 2 years 5 months, the upper teeth were for the most part carious, but in one of the patients the lower teeth were equally bad. About the effect upon the permanent teeth I know nothing, and should be glad to hear the experience of others.

Much less important than the influence of Brophy's operation upon the anterior part of the dental arch is its effect upon the width of the arch. Brophy proceeds upon the assumption that in normal infants the upper and the lower dental arches are equal in width; that in cleft palate the upper jaw is wider than the lower by the width of the cleft,

and that the condition of cleft palate is therefore not due to arrest of development, nor associated with any absence of palatal tissue. These assumptions are incorrect, as I have shown in a recent Hunterian lecture. And the further conclusion, that the proper treatment of a case of cleft of the hard palate is to squeeze the two maxillæ into contact with one another, is therefore ill founded.

A series of measurements shows that in normal infants the width of the upper jaw, measured between the outer sides of the alveolar processes, is greater by a few millimetres than the corresponding width of

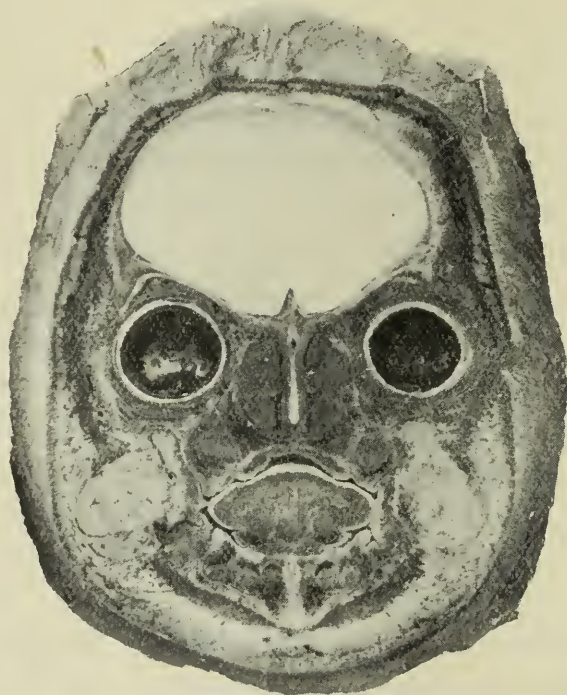


FIG. 1.

A section of the head of a normal foetus at full term. The upper jaw is seen to be wider than the lower.

the mandible (fig. 1); in bad cases of cleft palate this difference in favour of the upper jaw is greater, but is not as a rule enough to account for the width of the cleft. The explanation probably is that when there has occurred an arrest of development leading to cleft palate, the cleft is made wider by the two maxillæ being more or less pressed asunder. Forcible approximation of the maxillæ would result in an upper jaw narrower than the normal, and in many cases actually narrower than the mandible. But since Brophy's operation does not bring the maxillæ together, and does not bring them even greatly nearer to

one another, discussion of its effect in narrowing the dental arch is unimportant.

Other operations which directly attack the dental arch are those which are undertaken for the reposition of projecting premaxillary bones. In the case of an infant with a wide complete cleft, associated with hare-lip, no one could fail to be struck by the deformity of the premaxillæ, and with the wide gap at the anterior part of the dental arch; but in reading directions for the performance of Langenbeck's operation for cleft palate one is equally struck by the paucity of references to



FIG. 2.

Fig. 2.—Complete cleft palate (associated with left-sided hare-lip) in an infant aged 3 months. Note the bent nasal septum and the tilting of the premaxillæ to the right; also the cleft in the alveolar arch.



FIG. 3.

Fig. 3.—The palate of a boy, aged 15; a left hare-lip had been closed in infancy. The original cleft in the alveolar arch is now represented only by a groove (compare with fig. 2). This cleft was completely closed by one operation by Langenbeck's method.

methods of closure of the gap in the arch. The reason is a simple one. If one will operate upon the hare-lip soon after birth, in the vast majority of cases the growth of the parts, together with the pressure of the closed lip, suffice to bring the displaced premaxillæ back into

position, and so to close the defect in the arch without any further operation in the course of a few months. Sometimes actual union appears to have occurred; in other cases close apposition without real continuity (fig. 3). But occasionally the defect in the arch is not closed in this way.

I would refer once more to Brophy's operation, to point out that this is my main reason for thinking it generally unjustifiable. The alveolar arch can be restored without this severe procedure, and its performance accomplishes little if anything more. I admit, however, that reposition and wiring of the premaxillæ may cause a more exact restoration of the arch than would otherwise result. But such reposition, if carried out as a routine practice, will sometimes be overdone, and will lead to that very ugly underhung appearance which is by no means rarely seen, besides causing a faulty direction of the incisor teeth. On the whole, I believe that it is best not to do much in the way of reposition of the premaxillæ, except in extreme cases of deformity, but to unite the lip over them and leave nature to do the rest.

It is scarcely necessary to mention removal of the premaxillæ, a procedure which formerly held a well-recognized place in the treatment of hare-lip and cleft palate. I have seen patients upon whom it has been performed (fig. 4). They exhibited a gap at the front of the palate, a gap which sometimes nature had attempted to close by the drawing in of the alveolar processes of the two maxillæ; in this way the dental arch remained no longer an arch, but had become a triangle, with its apex in front. Nowadays, I suppose that the operation is seldom performed; it is doubtful whether it is ever justifiable.

Another procedure, of the results of which I know nothing, consists in scooping out the incisor teeth from premaxillæ which are much displaced forwards. It is difficult to see how this can facilitate reposition of the bones, but probably some who are present have seen the results and can say what the effects upon the dental arch may be.

(II) Among the second class of operations, those whose influence upon the dental arch is indirect, the most important are: (1) The turn-over flap operation (of Sir Arbuthnot Lane), and (2) Langenbeck's operation.

In neither of these operations is there any attempt directly to influence the shape of the dental arch, or directly to obliterate the gap between the bones, as there is in Brophy's operation; both aim at bridging the cleft by flaps consisting of soft parts alone. In the great

majority of cases in which these operations are performed, no effect upon the dental arch is to be observed, at any rate as regards its shape, but sometimes the effect may be considerable. The turn-over flap operation is the more likely to have an effect upon the arch, both because of the early age at which it is performed (seven hours is the youngest I have heard of) and as a result of the method of its performance. In this operation, for the purpose of closing a cleft in the hard palate (the only part which concerns us here), a flap is made, consisting of the whole thickness of the soft parts; the flap hinges at the edge of the cleft, across which it is turned; its raw surface is partly covered by the muco-periosteum at the opposite side of the cleft, and is in part left



FIG. 4.

Complete cleft palate in a boy, aged 5. There had been originally a double hare-lip; in operating for this in infancy the premaxillary bones had been removed.

uncovered; the surface from which the flap is raised is left bare. The contraction of scar tissue which results in this situation may either draw in the alveolar arch on that side, or (I do not know which) prevent its normal expansion; the result is the same in either case, the arch on the side from which the flap has been taken may be straighter than on the opposite side. And, with or without straightening of the alveolar arch, some of the teeth may become displaced inwards by this contraction. Irregularity of the teeth is of course a frequent accompaniment of cleft palate, quite apart from operations, but it generally affects the

anterior teeth. This post-operative displacement is something different, it often affects the premolar or molar teeth, and the displacement may be seen to be opposite a part of the palate which shows special evidences of scarring.

Apart from displacement, it is certain that the turn-over flap operation often causes injury to the temporary teeth, because if the cleft is so wide that the flap would otherwise not be broad enough to bridge it, the latter is taken not only from the palate, but also from the alveolar process, or even from the mucous membrane of the cheek; no doubt it is the raising of the flap from the alveolar process in such cases which damages the teeth. They are very commonly carious.

The plan of Langenbeck's operation is different. On each side of the cleft the muco-periosteum is raised from the bone in the whole breadth of the hard palate. This is done, according to the taste of the operator, either by an elevator introduced at the edge of the cleft and working outwards, or by a similar instrument passed through a short incision at the outermost part of the hard palate, close to the alveolar process, and working inwards. The flaps, when freed, have their edges pared, and are then sutured in the middle line. Owing to the height of the palatal vault they are nearly always broad enough, in the case of the hard palate, to meet without any tension, even when no lateral incisions have been made. It must be noted that the flaps are often raised not only from the palatal process but also from the inner aspect of the alveolus; it is the degree of development of the alveolar process which determines the height of the palatal vault, and not the direction of the palatal processes; the latter are always horizontal, whether the arch is high or low.

After an operation of this kind, cicatrization occurring in the space between the flap and the bone from which it has been raised may occasionally lead to drawing inwards of a tooth or teeth. I think this is decidedly rare. I know nothing of flattening of the alveolar arch as a result of Langenbeck's operation, and shall be glad to hear if it is within the experience of others.

DISCUSSION.

Sir ARBUTHNOT LANE said he was glad the Section had done him the honour of asking him to be present, as he had enjoyed listening to Mr. Blakeway's interesting paper. In determining the course of treatment to be adopted in cleft palate, one ought to decide in the first instance on the general principles that should govern it. Many years ago he worked at the mechanics of the bones of the face and came to the conclusion that the factors determining their development were three in number. The most important, and the one that came into action immediately the child was born, was the pressure exerted upon the sides of the nasal cavities by the air during the process of respiration. This affected the size of the nasal cavities and of the cavities that communicated with them, and so influenced the height of the palate and the breadth of the alveolar arch, as well as the shape of the face. It was hardly necessary to call attention to the value of a well-developed nasopharynx to the individual both from the point of view of appearance and health as well as of speech. The more perfect the development of the nasopharynx the finer was the face, the more healthy and robust the individual and the more perfect the voice. The tongue was an associated factor which played the chief part in the development of the lower jaw. It also affected the form of the palate and the alveolar arch of the upper jaw. The teeth became a factor at a later period in the life of the infant, both in occupying space and transmitting pressure. The importance of the teeth as a factor in the evolution of the bones of the face was very familiar to the dental surgeon. When he came to consider the treatment of clefts of the hard palate, which at that time was never undertaken till the child had reached the age of 3 or 4, he realized that owing to the communication which existed between the nasal cavities and the mouth, the pressure which was in normal condition exerted on the sides of the nasal and associated cavities by the forcible transmission of air through these spaces was practically in abeyance during the first four years of life. The absence of this pressure resulted in a diminution in the size of the nasal cavities both vertically and laterally, in a progressive narrowing of the cleft and in rendering the segments of the cleft more vertical. This imperfect development of the nasopharynx in a case of cleft of the hard palate which had reached the age of 4 was very advantageous to the surgeon, who then operated by approximating the edges of the elevated muco-periosteum, since it facilitated that procedure very materially at the cost of the development of the nasopharynx and other structures which depended on it for their perfect evolution. That was evidently the reason for the delay in operating at that age. At that time an operation on a child a year old was deemed to be impossible. He found that by employing the flap method he was able to close the largest clefts in the youngest infant, some of which, especially the horse-shoe variety, could never be closed except by this method, and then only before the temporary teeth had erupted. The earliest age at which he had operated was 7 hours,

but he had closed the cleft in the palate of a child removed by Cæsarean section at the age of 7 months. He made it a rule to operate in cases of complete cleft of the hard palate as early in life as possible, and upon the soft palate at a convenient subsequent date. In the case of cleft of the soft palate where the hard palate was complete, there was not the same importance of operating at a very early date, since the mechanical factor exerted by air pressure was not in abeyance. A circumstance that favoured early operation was that Sir Almroth Wright had shown that while the resisting power of the tissues of the infant was equal to that of the mother at birth it gradually diminished during the first month of life. Another factor influencing early operation was the fact that children with complete clefts of the hard palate had, as one would expect, much less vitality than the normal infant, and their mortality was correspondingly greater. Those who resided in a children's hospital and those who performed a very large number of operations on these cases of cleft palate were very familiar with this fact. This had been denied by those surgeons who preferred to postpone operation and who consequently operated on the more robust specimens only, the more feeble and the more deformed having previously succumbed. Even after the cleft in the palate had been closed the vitality of these children was somewhat lower than in the normal child whose intelligence was usually of a distinctly higher order. The vitality of the cleft palate infant in the class who filled their children's wards was also influenced by the greater difficulty which was experienced by the parents in feeding the child. In some cases the abhorrence of the parent for the child's deformity and feebleness of physique often resulted in neglect of the child. These children not infrequently suffered from other faults of development. On all these grounds the importance of early operation was indicated. Since early operation was, like all other innovations in surgery, which was practically a creed and not a science, opposed for many years, he was able to operate on as many cases as he pleased, as he was flooded with material. As many as 125 cases were frequently on the books waiting for admission. Recently other surgeons had commenced to operate earlier in the life of the child, so that the supply of cases had diminished, being limited to the more severe types of the deformity. It was probable that no surgeon would again ever perform so many operations of this kind, since the peculiar circumstances could never recur. He was more and more assured of the advantages which operations performed in early infancy afforded, and this opinion was now shared by a large number of surgeons. In an ideal case the cleft or clefts of the lip and hard palate would be closed at birth and that in the soft palate before the end of the first month. The changes that ensued in the first month after early operation were very great and this was much more marked in the first week, less marked in the second, still less in the third, and still less in the fourth week of life. The advantage of early operation was now generally accepted by surgeons all over the world, and the old rule of delaying operations till the fourth year was limited to only the conservative few. He divided the treatment of cleft palate under three heads, all of which

were of equal importance: First in order, surgical interference. Secondly, the instruction of the child in voice production. One of the most striking results of this treatment was to show that the benefit which was obtained from skilled tuition bore no such very close relationship to the perfection of the surgical result as one would expect. Originally, before he took up the question of the treatment of cleft palate by early operation, the child had already learnt to speak by the time the cleft was closed, which was certainly after three years and probably later. The surgeon, therefore, did not expect that his operation would remedy the defective articulation, and apparently his procedure was undertaken to improve only the appearance and comfort of the patient. It was owing to the absence of the educational portion of the treatment that children operated on in the children's hospitals suffer most. Being operated on early in life and being scattered at once all over the country, they had no means of teaching them to speak, nor had they been able to devise any except in a few cases. He asked Mr. Cortlandt MacMahon, who had devoted a great deal of time to many of his cases, to let him know how children operated on at an early period by this method spoke if properly educated in speech production, and the following was Mr. MacMahon's reply: "With one exception in a child whose intelligence was distinctly below the normal your cases have made good progress. Some speak almost perfectly." The importance of speech education could not be exaggerated, and he was very glad to have the opportunity of acknowledging his indebtedness to Mr. MacMahon for his great skill and patience in this form of treatment. The third share, and by no means the least important in the treatment of these cases, was that undertaken by the dental surgeon, whose treatment commenced as soon as the teeth afforded him a secure grip for his apparatus. Not only did he arrange the teeth in a symmetrical series and affect the outline of the alveolus, and so improve the appearance of the individual, but he also enabled the patient to make many sounds which would otherwise be impossible if the teeth were displaced. It had been asserted that the flap operation produced a scarring and cicatrization of the roof of the mouth with displacement of the teeth. He did not believe there was any evidence whatever for this statement. He believed that this fallacy had arisen from the fact that the flap was almost always taken from the smaller segment of the palate whose alveolar margin at the time of operation was already well within the normal limit. The position had changed materially since he first studied and formulated the general principles that should guide them in the treatment of cleft palate. These principles had been accepted generally, as evidenced by the fact that surgeons were now operating as early in life as the particular method they favoured enabled them to do. At the present moment the discussion was limited to the consideration of the best form of operation, and surgeons were much divided on this point. Time and experience alone would settle this question. To a large extent the procedure favoured must depend on the degree of the deformity and on the skill and ingenuity of the operator.

Mr. WARWICK JAMES said he had been working on cleft palate cases for over ten years ; but he had difficulty in being able to produce results during that time, as the patients were usually seen at an early age, and the treatment could not be regarded as complete until the patient had reached adult life. He had had two patients whom he could describe now as adults, and he exhibited models and photographs of them and some others, his object being to show the deformities which occurred in the dental arch. These deformities occurred in all three planes—displacement occurring inwards, backwards, and in a vertical direction. In cases in which cleft palate had been operated upon there was always some degree of deformity of the dental arch, and practically always in the three planes. He had not always known in his cases by what method the operation had been performed, but he had seen cases at the Royal Dental Hospital, Great Ormond Street Hospital, and privately, which had been dealt with by various operators. The illustration of a case aged over 20 showed the condition prior to and after treatment, demonstrating the very considerable change which could be effected. This case showed the movement which had been effected of the bony structures as well as the teeth. In correcting the dental arches several objects would have to be achieved. The arch itself would need to be corrected so that it approximated as far as possible to what would be a normal condition. If this was achieved the appearance of the patient would be greatly improved, the teeth would be rendered more serviceable, and the possibilities of improvement in speech greatly enhanced. Other benefits would accrue, such as greater mobility of the lip; and in fact, many of the great discomforts from which such patients suffered would be removed. After the correction of the teeth in their alignment it would probably be necessary to build up the teeth in order to correct the mal-occlusion. He had dealt with cases both in hospital and private practice at the age of 2 years and upwards. The great difficulty in dealing with hospital patients rendered their satisfactory treatment almost impossible. The two cases shown where treatment had been completed were seen privately. He could not discuss the question from the surgical point of view, as he had not operated upon cleft palate. With regard to the deformities which occurred in the dental arch, he was of opinion that they were dependent upon two factors. One was the growth of the jaws being impaired; the other, the normal forces which moulded the arches—namely, the tongue, lips, and cheek being abnormal in their action, particularly in those cases where the habit of mouth-breathing had been established. He thought that the latter factor largely accounted for the inward displacement. It was necessary for the dental surgeon to remember that the nasal cavities were frequently undeveloped, and in carrying out correction of the arches this condition should be improved as far as possible, and restoration of nasal breathing brought about. The ordinary processes in the production of irregularity in the teeth might co-exist, but he did not consider them of great importance, as they could be corrected in the ordinary manner by the methods usually adopted. Sir Arbuthnot Lane had spoken of the condition being dealt with after the eruption of the permanent teeth. An attempt to treat the cases

could be made at an earlier stage than that, but on account of the flatness of the arch it was often difficult, and in consequence it was not easy to use an appliance. Moreover, the introduction of a fixed appliance took time, and these children were found to be peculiarly shy, owing to the previous operation. After eruption of the permanent teeth treatment was easier, as the parts had developed. In conclusion, he stated that the possibilities of a good result being obtained might be regarded as most promising.

Mr. THOMAS H. KELLOCK said that if he accepted the President's invitation to make some remarks, they must be from the point of view of the operating, rather than that of the dental, surgeon. He need not apologize for disagreeing with the views of Sir Arbuthnot Lane. He differed from him as to the age for operation and the method of operation on children with cleft palate. It had been said that a large proportion of these children died if left alone: that they neither breathed nor fed properly, and, in the end, succumbed to their deformity. Some years ago he found that at Great Ormond Street Hospital nearly all the cases of cleft palate were being operated upon as babies; and some of his colleagues and himself, who preferred to leave the operation until the child was a little older, had few of these cases coming under their care. He protested that the palates of these babies were, from his point of view, being spoiled at an early age. He therefore, as it were, bespoke some of them, which were kept for him until they were older. When he got them they were a year or 18 months old, and he was surprised to find what remarkably fine, healthy children the majority of them had become. It was true that in the interval a certain proportion of them might have died from inanition. It was easy to prove that if these cases were operated upon a certain proportion of them died; but it was difficult to demonstrate that if operation were not done they would likewise die. His contention was, that if one were going to get an ideal result in a child's mouth, that result could best be secured by a median-line suture of the palate. Mr. Blakeway had shown that when a flap of muco-periosteum had been taken almost entirely from one side of the hard palate, it would bring about an inequality in the size of the two sides of the upper jaw. If the contraction following the operation for cleft palate were going to displace the teeth even slightly, it was far better that such displacement should be symmetrical, rather than that one side should be drawn in, and the other left in its former position. Therefore, if he were asked when he preferred to do an operation for cleft palate, he would reply, at the earliest age at which he could reasonably hope to derive success from a mid-line suture. He did not think the operation was much more difficult then, and if there were a good result with healing of the palate in the middle line, that result was the best that could be obtained. What was the result of the flap operation in the case of the little baby? If the child were 8 to 10 years old, it would be found that the palate was drawn over to one side, and the soft palate was likewise displaced, scarred, and tightened; also, articulation was very

deficient, and one found the soft palate stretched across the back of the mouth almost like the string of a bow and leaving a large gap between it and the back of the pharynx; it did not move properly, and the speech was very bad. If the child was to have good speech it was essential to ensure a movable soft palate. The ideal time and operation depended somewhat on the width of the cleft and the height of the palate, so keeping up the symmetry of the palate. With regard to the subsequent displacement of the permanent teeth, he thought this was less and less asymmetrical in the middle line operations than in those where a flap was turned across.

Mr. BLAKEWAY, in reply, said that he supposed all would agree with Sir Arbuthnot Lane that these children ought to be operated upon as soon as possible after birth, if by such an operation the best possible result was thereby attainable. But he felt sure that the children who were operated upon at the early age mentioned by Sir Arbuthnot Lane did not obtain the best results. He supposed that the most important good which one hoped to do for these children by operation was to improve their speech—that was of great importance in their after-life. Owing to the kindness of Sir Arbuthnot Lane, he had been able to trace a number of cases which had been operated upon by the flap method, and to publish the results. Of 100 consecutive cases, he had succeeded in tracing eighty-seven, at periods up to nine years after operation. The result was very striking. Among the cases of complete clefts, in both hard and soft palates, only one of the cases he saw—and he was only speaking of cases he saw—showed a good result as regards speech, and that child had not been operated upon at the very early age which had been advocated; he was aged 14 months when the operation was done, not far short of the age at which Langenbeck's operation was usually performed. The vast majority of the children who spoke badly in after years had been operated upon at an earlier age than 6 months. He considered the time had come when one ought not to argue, from theoretical considerations as to the growth of the palate, as to what kind of operation should be performed. Surely this was the kind of case which was amenable to the statistical method; and if the late results of one form of operation done at a certain age were found to be the best, the operation at that age and that form of operation should be persisted in, whatever theoretical considerations were entertained.

He had been much interested in the casts exhibited by Mr. Warwick James, because they were not the kind of cases which usually came his (the speaker's) way. He assumed that most of them had been operated upon by the flap method. He thought it was unusual to see cases which had been operated upon by Langenbeck's operation presenting such a deformity of the dental arch—it was so in his experience.

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PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE EIGHTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1914-15

SECTION OF OPHTHALMOLOGY



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1915

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SECTION OF OPHTHALMOLOGY.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Section of Ophthalmology.

November 4, 1914.

Mr. PRIESTLEY SMITH, F.R.C.S., President of the Section, in the Chair.

Dilated Pupil of Argyll-Robertson Type in a Young Girl showing well-marked Contraction to Closure of the Lids.

By A. HUGH THOMPSON, M.D.

M. H., A STUDENT, aged 16, a somewhat anæmic but otherwise healthy girl. In February, 1913, I tested her refraction, and the left pupil was then normal. It was noticed by her mother to be enlarged in the following July. In May, 1914, I saw her again, and the condition has remained unchanged since then. In moderate light the left pupil is dilated to between two and three times the diameter of the right. It does not react to light either directly or consensually. It does contract well to convergence, though somewhat slowly. It also shows a marked contraction immediately after a voluntary closure of the lids. Vision is not affected either for distance or near. Right eye normal. Fundi normal. Refraction: R., $-0.75D. \frac{6}{8}$, J.1; L., $-0.25D. \frac{6}{5}$, J.1. The knee-jerks are normal.

Case of One-sided Internal Ophthalmoplegia.

By A. HUGH THOMPSON, M.D.

J. B., AGED 28. Two years ago this patient was invalided from the Navy on account of a supposed aneurysm, but the only time when he has experienced any loss of bodily strength was during his enforced rest in a hospital. He was formerly a stoker in the Navy and is now

2 Thompson: *Case of One-sided Internal Ophthalmoplegia*

doing similar work in a brewery. The right pupil is dilated to 6 mm. diameter, and barely reacts at all either to light or convergence. Accommodation is also lost. The condition has persisted unchanged, according to patient's account, for over two years. The right pupil began to enlarge three years ago. Patient had syphilis at the age of 22, for which he had 120 mercurial injections. Fundi are normal. Knee-jerks normal. Vision: R., $\frac{6}{12} + 1.0 \frac{6}{5} + 4.5$, J.1; L., $\frac{6}{5}$, no Hm., J.1.

Dr. ROCKLIFFE said he remembered two cases, one of which was at the present time under observation, of mydriasis and irido-cycloplegia; the other a man whom he saw twenty-five years ago. The present patient was a woman, aged 36, with full persistent mydriasis of the left eye. He first saw her on July 31 last, when she had had the condition for a fortnight, with headache. Her vision was absolutely normal, though she was slightly hypermetropic in both eyes, but the left eye required +3 to read J.1. There was a strong family history of retinitis pigmentosa, but no retinal or pigmentary change, the fundi being only slightly mottled at the periphery, and nothing abnormal could be discovered except a dilatation of the pupil. She had now been under his observation three months, taking $\frac{1}{16}$ gr. of mercury t.d. This was stopped, on August 26, on account of colic, when Easton's syrup and eserine drops, together with + cylinders, were prescribed. Three months later he again put her on hydrargyrum, and when he saw her that morning, beyond that her headache had been relieved by the spectacles, she was in the same condition. The point he wished to emphasise was that the eserine was beginning to lose its effect, and that to-day the pupils are only semi-dilated. The other case he reported in 1888 (*Ophthalmological Society's Transactions*¹). The patient was a young man, a clerk in a shipping office. He was first seen in 1886 with left irido-cycloplegia only. His vision was $\frac{6}{6}$ and J.1. Field N. The case was shown more especially as exemplifying the great tolerance of some people to potassium iodide and hydrargyrum. The subsequent history was of interest. Fifteen months later he had paresis, R. VI, with full mydriasis of both. Accommodation: R.E., normal; L.E., 6 in. No other symptoms of cerebral, spinal, or rheumatic disease, tuberculosis, or of syphilis could be detected, and he was ordered potassium iodide, blister, and, later, galvanism and eserine drops. Three months later (November, 1887) *neither pupil contracted under eserine*. Ten days later, complete iridoplegia in both, with diplopia, had set in. Mr. Nettleship saw him in January, 1888, and diagnosed the affection "most likely central and syphilitic," and advised increasing doses of mercury. During the period of two months he took $\frac{1}{2}$ lb. of potassium iodide, and during the four months $1\frac{1}{2}$ oz. of pure mercury, without any poisonous symptoms, except slightly spongy gums, and there were no material alterations in his symptoms, with the exception

¹ *Trans. Ophthal. Soc. U.K.* (1887-88), 1888, viii, p. 258.

that his pupils now again responded to the *eserine*. The treatment was then stopped, and four months later (September, 1888) he recommenced potassium iodide and hydrargyrum. The following month (October) the left pupil was almost normal, the right distinctly improving, no strabismus and very slight diplopia. The patient was not then seen for eleven years until 1899, when he had been married six years: one stillborn child, no miscarriages. He had then sudden mistiness of distant and near vision. R.V., $\frac{6}{12}$; L.V., $\frac{6}{18}$; right pupil 9 mm. and left pupil 6 mm., with some doubtful hæmorrhagic remains above the R.O.D., and he was ordered iron and atropine drops for further examination. He (Dr. Rockliffe) never saw the man again, but heard from a friend in his office that he gradually became incapable of doing his work, and unfortunately an inebriate, and died June 11, 1911—i.e., about twenty-eight years from the first symptoms—in the Willerby Asylum. Dr. Anderson, the medical officer, had reported only that morning that he died from general paralysis, and the post-mortem revealed a typical general paralytic brain, with no growth or local lesion. The question arose, was the *varying effect of eserine* on the pupil in each case a *coincidence* or *symptomatic* of general paralysis?

Case of High Myopia in an Infant; Hereditary and Congenital.

By N. BISHOP HARMAN, F.R.C.S.

THE child, Edward M. (Generation IV, 5), was seen in April of this year (1914), when aged 9 months. The mother thought there was something wrong with his eyes. When examined under atropine retinoscopy showed an error of refraction amounting to $-16D.$ in the vertical meridian, $-14D.$ in the horizontal. The myopia is evidently axial; nothing abnormal can be made out in the refractive media. The fundus of each eye is thin, and the retina so little pigmented that the whole of the choroidal vessels can be traced. There is no stretching about the disks. The child is a nice, chubby baby, fair-haired, with irides slightly tinged with brown.

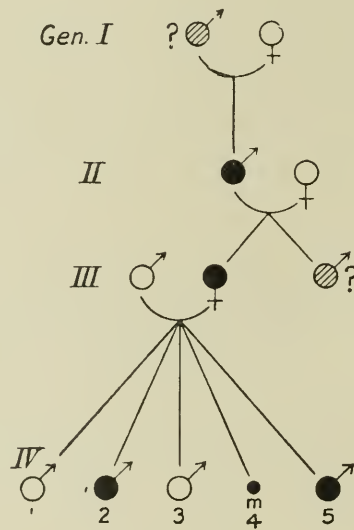
The family history has been traced into four generations, with the following result:—

Generation I: Male, unknown. His wife is known to have been very long-sighted.

Generation II: One child only, a male, who was very short-sighted. He worked as a plasterer. He married a woman whose sight was good. She died in childbirth at the age of 25.

Generation III: Two children, offspring of the foregoing. The elder, a female, was very short-sighted as a girl. At the present date she is aged 40, and has a cataract in the right eye, with poor projection. The left eye had been excised on account of "internal injury caused by a blow" at the age of 17. The second child was a boy who died in infancy, condition of eyes unknown.

Generation IV: The children of Generation III. (1) This woman married a normal-eyed man, and had five pregnancies: (1) Harry, aged 8; dark hair, dark brown irides, retinoscopy +0.5D. sphere, fundus normal. (2) William, aged 6; dark brown hair and irides, retinoscopy



Pedigree of family with congenital high myopia.

in right eye $-18D.$ vertical, $-16D.$ horizontal; left eye $-14D.$ vertical, $-9D.$ horizontal, very thin fundus, pigmentation irregular. (3) John, aged 5; fair hair and blue eyes, retinoscopy $+0.5D.$ cylinder, axis vertical. (4) Miscarriage. (5) Edward, aged 9 months, whose condition of high myopia is noted above.

Mr. HARMAN, in answer to the President, said there were no fundus changes. Hypermetropia of higher degree had been recorded, but he did not know of a case of higher degree of myopia in so young a child.

Tumour Lymphangiectasis of the Lower Lid.

By WALTER H. JESSOP, F.R.C.S.

H. D., FEMALE, aged 51, was admitted into St. Bartholomew's Hospital with a swelling about the size of a small orange depending from the lower eyelid of right eye. Two years ago, after a hard day, scrubbing



Lymphangiectasis of lower lid.

floors, she noticed a swelling on the right eyelid, like "a small bladder of water." The swelling has gradually increased. About three months ago it increased much more rapidly and attained its present size. Her doctor lanced it and let out a little watery fluid. She thinks the swelling was a little smaller after the lancing, but quickly resumed its former size. The tumour has never been painful; there is no history of a blow or of any disease, such as erysipelas. She has been married

thirty-one years and had eight pregnancies, five children being alive; two children dying in infancy and one miscarriage.

The tumour hangs from the lower lid a very little distance from the ciliary border. It appears to arise from the whole length of the lid and has therefore a broad skin pedicle. It is covered over by the skin, which is smooth from stretching. It is globular in shape, 4.1 cm. transversely, 4 cm. vertically, and 1.7 cm. antero-posteriorly. The tumour pits very easily on pressure, and is in most of its extent semi-fluid to the touch; in the lower part it is somewhat thickened. It is transparent by transillumination. On the conjunctival surface of the lower lid there is a bluish, transparent appearance. The swelling was punctured by the needle of a syringe, but no fluid could be drawn off. On moderate pressure the tumour does not become smaller.

During the last few days the right upper lid has been swollen and œdematous, and in the right temporal region there has been some thickening and œdema. The bridge of the nose is flattened, and on both sides between the nose and the eyelids the skin is thickened and œdematous. Skiagrams taken from the side and in front show no changes in bones or sinuses. Vision in both eyes $\frac{6}{6}$, and the ophthalmoscopic appearances are normal. Mr. Harmer examined the throat and nasal passages but found nothing abnormal.

I have never seen anything exactly like this condition, but have found two recorded cases resembling it: one described by Teillais in 1882, both upper lids being involved; and the other by de Wecker in both lower lids. The condition was originally called elephantiasis nostras. Evidently it was of the nature of a lymphangioma.

After-treatment.—Under a general anæsthetic considerable pressure was applied to the tumour, when it became smaller, and on continued pressure the swelling disappeared, leaving only the skin surfaces. At the same time there was much swelling in the temporal region; the eye was greatly proptosed, the conjunctiva chemosed, and the upper lid swollen and œdematous. The tumour regained its original size after six hours. On November 24, under a general anæsthetic, the tumour was removed by an anterior and posterior flap, and the skin surfaces stitched together, leaving in a drainage-tube at the nasal side. On cutting into the tumour the skin surface was thickened and a cavity in the centre lined by endothelium contained blood-stained serous fluid. This endothelial lining extended up to beneath the conjunctiva of the lower lid, but there was no communication to be found by probe with the temporal region. On pressing over the œdematous swelling in the

temporal region blood-stained serum exuded. The tumour, therefore, was formed by a cyst-like cavity pushing down and stretching the lower lid. The wound healed up quickly, but there is still some swelling in the right temporal region beneath the fascia.

Professor Andrewes kindly examined the specimens and reports: Section shows that there is no new growth. The condition appears one of lymphangiectasis, pure and simple. In the loose connective tissue of the dermis are dilated lymph spaces, irregular and ragged, not lined by endothelium. The skin is otherwise normal. The fluid is mixed with a good deal of blood, but has a much lower protein content than pure serum—2.17 per cent. It is therefore probably a mixture of lymph and serum.

From the anatomical and pathological evidence the tumour was evidently caused by lymphangiectasis, the obstruction probably being in the orbit or temporal region. The enlargement of these spaces in the lower lid structures produced a large cystic formation which, gradually dilating, formed the dependent swelling of the lower lid.

DISCUSSION.

Mr. LESLIE PATON said he had seen a case similar to Mr. Jessop's, where the causation was fairly obvious. There had been severe lupus, and the scar tissue was adherent to the bone and had caused complete blockage of the lymphatic ducts draining the eyelid. It was a simple œdema, and therefore the term "elephantiasis" was almost justifiable.

Lieut.-Colonel ELLIOT said he had seen a similar case in India, of which he published the notes in the *Ophthalmoscope* of August, 1912.¹ It was examined and was found to be due to *Rhinosporidium kinealyi*. The growth was excised with good results.

Mr. BISHOP HARMAN said that a case was shown some years ago before the Laryngological Section by Dr. Davis,² in which the upper maxilla had been removed because of new growth. After the operation there was a condition of the same type though not so extreme as this. In that case he (the speaker) implanted long silk drains reaching to the side of the neck, and the effect was fairly good, even though the threads had to be bent at a right angle over the site of the primary operation scar. The patient was now dead.

¹ *Ophthalmoscope*, 1912, x, p. 428.

² *Proceedings*, 1911, iv (Laryng. Sect.), pp. 30, 86.

Case of Confluent Tubercle of the Iris.

By H. L. EASON, M.S.

A. T., AGED 8. On September 12, 1914, the boy's parents saw a white spot on the iris. Three days later they brought him to Guy's Hospital. At that time there was a smooth, oval, white mass, in shape and size resembling an ant's egg, protruding from the anterior surface of the iris on the outer side, about half-way between the pupillary margin and the angle of the anterior chamber. The pupil was active, there was no sign of any inflammatory reaction, and vision was $\frac{6}{9}$. The disk and fundus were normal and there were no vitreous opacities.

At first the mass grew very slowly, but the eye gradually became inflamed. By October 10 the growth had entirely filled up the angle of the anterior chamber for about one-fifth of the circumference, and extended as far as the pupillary margin. There was also very definite vascularity of the lower and outer corneal limbus and the surrounding conjunctival tissue. On this day an injection of 0.0002 mg. of new tuberculin was given by Dr. Eyre. After October 20 the mass developed rapidly. Several secondary nodules appeared on the anterior surface of the iris, the original deposit increased very much in size, and there was copious white confluent keratitis punctata. On October 26 the growth commenced to perforate the globe in the ciliary area, and the infiltration of the sclerotic in that region proceeded rapidly after that date. Further injections of new tuberculin (0.0002 mg.) were given on October 22 and November 1 with no perceptible effect upon the progress of the disease.

Mr. EASON added that the case was evidently not improving with tuberculin and he imagined that enucleation was now indicated. He showed the case because it was his impression that such conditions were comparatively rare; he had only seen one other in ten years. His previous case perforated more rapidly than this—viz., in about three weeks. Other members of the Section, however, considered such cases fairly common.

Section of Ophthalmology

**Multiple Cysts in the Anterior Chamber derived from a
Congenital Cystic Growth of the Ciliary Epithelium.**

By W. T. HOLMES SPICER, F.R.C.S., and
R. AFFLECK GREEVES, F.R.C.S.

(I) CLINICAL HISTORY OF CASE, BY W. T. HOLMES SPICER, F.R.C.S.

A. W., AGED 16 months, was brought to Moorfields on January 8, 1907. Nothing had been noticed about his eyes till he was 3 weeks old, when he was seen to squint. This called attention to his eyes, but



FIG. 1.

Three finger-like bodies protruding into the anterior chamber from behind the iris. Local opacity of lens.

nothing else occurred till he was 3 months old, when a white spot appeared in the eye. This has steadily grown since. He was born at full time, a healthy child; no instruments were used and the birth was easy. He has never had any injury. Has never been out of England. At the time of first appearance of the growth he had had no meat, and also had had no contact with dogs, although one or two were kept by his parents. Nothing had occurred in his life to suggest a parasitic infection. There was no family tendency to any constitutional disease or peculiarity, as far as could be ascertained.

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On January 8, 1907, when he was first seen, he was a remarkably well-nourished, healthy, and well-formed child. He resented interference, but it did not seem that the eye was tender to touch. For further examination he was put under chloroform and was subsequently taken into the hospital. Under chloroform, the lids, cornea, and sclera were normal, there was no conjunctival nor ciliary redness, nor any discoloration of the ciliary region. The iris was of the same colour as its fellow, but the pupil was larger and less active. The lower part of the iris from about 5 to 7 o'clock was pushed forward, shallowing the anterior chamber to some extent; the tension was not raised. At this part of the pupil, corresponding to the displaced iris, there were

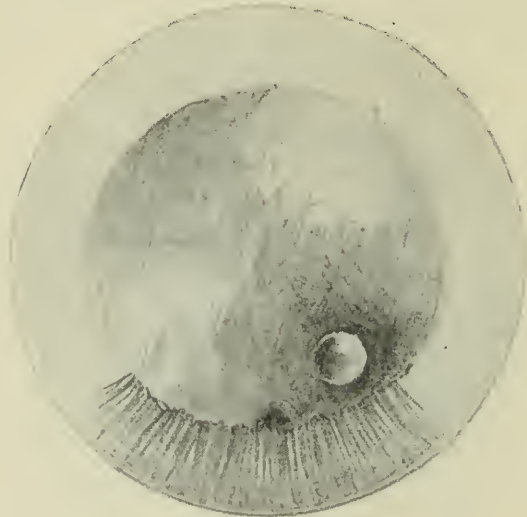


FIG. 2.

Shows the first cyst lying in the anterior chamber. General opacification and partial absorption of the lens.

three dirty white or buff-coloured finger-shaped projections coming from behind the iris; two of them just appeared in the pupillary area, a third, the middle one, turned forwards round the pupillary edge and lay on the anterior surface of the iris. They looked solid, and the largest of them seemed to have a darker centre, suggesting a blood-vessel (fig. 1). The lens was opaque at its lower part but quite clear above. The fundus could be seen, the disk and upper part were normal; the lower part could not be seen, and gave no fundus reflex, owing partly to the lens opacity and partly to the growth which seemed to occupy this region.

I was not familiar with the appearance and was glad to ask my

colleagues to see it. Opinions varied a good deal, tubercle and glioma were spoken of, but a majority thought there was no need for removal of the eye.

The next note was two years later, when the mass was thought to be shrinking.

In 1911 the lens had become generally opaque; there was no trace of the buff-coloured projections, but a broad posterior synechia marked their former situation. The anterior chamber was deep, and a grey,



FIG. 3.

Ciliary staphyloma above. Seven cysts floating in the anterior chamber, one having apparently two daughter cysts growing from it. Remains of other cysts (?) lying on the iris.

translucent, almost clear ball was floating unattached in the aqueous, moving with every movement of the eye (fig. 2). It could be examined thoroughly without difficulty, and no trace of structure could be made out, neither head nor hooklets nor anything to suggest its nature.

By the end of 1912 there were three of these bladders floating untethered in the anterior chamber, but seeming to hang together. At

the upper part of the eye the sclerotic was beginning to show signs of thinning just outside the cornea.

He was shown at this Section in the summer of this year. The eye had been getting painful for some months. There was discoloration of the ciliary region all round, with slight congestion and occasional straggling vessels. At its upper part there was a definite plum-coloured staphyloma with numerous large vessels running towards it; the pupil was dilated and the tension was raised. The anterior chamber was deep, with clear contents, and presented a most striking appearance. There were seven clear, transparent bladders or cysts floating in the aqueous (fig. 3). They moved freely and rebounded from each other or from the walls of the anterior chamber just like toy balloons, except the lowest ones, which were fixed. Each cyst had a thin wall, with structure enough to be just visible when looked at directly, but was chiefly visible by the reflections from its surface; where the cyst lay in front of the iris the colour of the latter could be seen through it. The cysts were of varying size, the largest was about 3 mm. in diameter, and there were two opaque, irregular white masses lying in the iris which looked like collapsed or dead cysts. No structure, nothing suggesting a head or hooklets, could be made out by the corneal microscope, but one of the cysts—the small one in the centre of the drawing exhibited—appeared to be throwing off two buds, and, perhaps, another one below was also budding. The two cysts which appeared to be budding were smaller than the others and suggested a loss of part of their contents to the daughter cysts (fig. 3).

As the eye had begun to be troublesome, consent was obtained for its removal, which was done this October.

(II) PATHOLOGICAL DESCRIPTION OF THE EYEBALL, BY
R. AFFLECK GREEVES, F.R.C.S.

There was a considerable general enlargement of the globe, in which, however, the cornea did not partake. Besides this, a marked local scleral ectasia was present in the ciliary region, up and out.

The eye was hardened in Zenker's fluid followed by formalin, and frozen and divided sagittally into two unequal parts in such a way that the section just opened the inner periphery of the anterior chamber. The coats of the eye were found to be in situ, the angle of the anterior chamber was completely occluded by a wide peripheral

synechia, and the papilla was deeply cupped. In the anterior part of the vitreous three small and apparently free cysts were found. The lens substance was almost entirely absorbed, the remaining capsule forming a diaphragm-like membrane. A number of small whitish bodies of various sizes, some rather larger than a pin's head and some smaller, were attached to the region of the root of the iris and ciliary processes round the whole circumference. Several cysts of various sizes were present in the anterior chamber, several of which were free. There was a colony of small cysts between the iris and lens capsule up and out in the neighbourhood of the ciliary staphyloma,

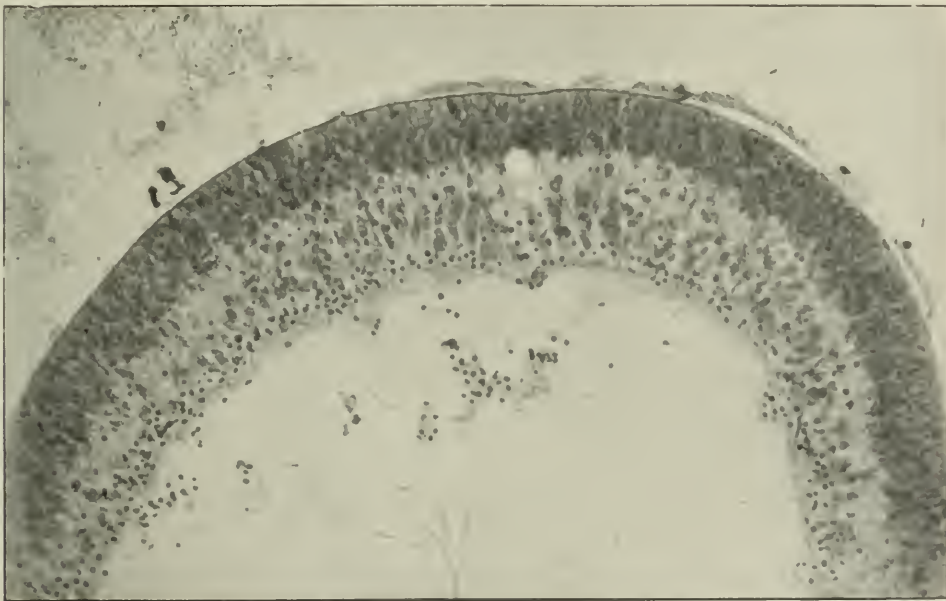


FIG. 1.

Section through wall of free cyst, showing structure resembling embryonic retina.

filling up the space between the back of the iris and the lens capsule. The retina looked normal, except for the presence of some small depressions here and there in the equatorial region.

Both parts of the divided globe were mounted in celloidin, and numerous serial sections were taken from different parts. Free cysts were removed from the vitreous and anterior chamber, and mounted separately, and separate sections of these were cut (fig. 1). The cyst wall in each case was found to be made up of cell nuclei contained in a delicate supporting substance, and these structures were of such a nature and arranged in such a way that the membrane forming the

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cyst wall bore a striking resemblance to embryonic retina at about the sixth or seventh week of foetal life. The structure was as follows: On the outer aspect of the cyst was a definite membrane, representing the external limiting membrane, and immediately beneath this a layer of densely packed oval nuclei with their long axes at right angles to the membrane. Many of these nuclei showed mitotic figures. Next to this layer came a zone of scattered nuclei tending to be rather rounder in shape, between which a delicate fibrillar stroma resembling neuroglia could be seen. In this layer were numerous small deeply staining

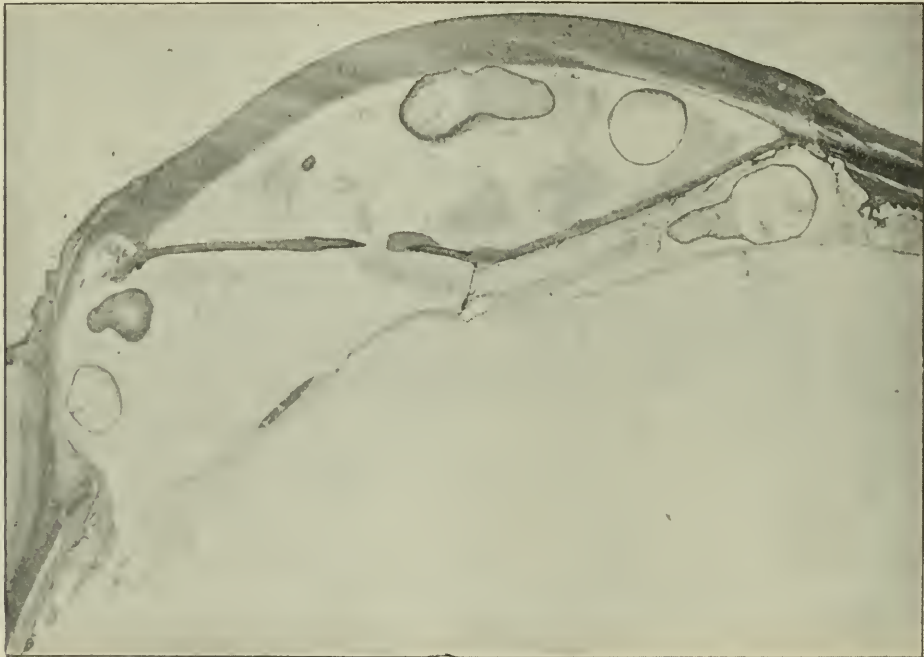


FIG. 2.

Section through anterior part of eye. The ciliary staphyloma, containing free cysts, appears to the extreme left.

degenerate nuclei, similar to those found in necrotic parts of a glioma. Internally, a very delicate membrane could sometimes be made out, but oftener the inner surface was irregular and covered with necrotic nuclei.

Sections through the entire globe (fig. 2) revealed the condition to be one of a new growth of the unpigmented layer of the epithelium of the ciliary body. On examining several sections it became evident that the place of origin of this growth was close to the junction of the root of the iris with the ciliary body, up and out, in the situation of the

ciliary staphyloma. Here, an erosion, and to a certain extent an invasion of the sclera by the growth, had taken place, with the result that the thinned sclera had given way, causing a separation of the site of the growth into two parts between which an intermediate staphylomatous area without epithelial lining had formed. Both layers of the normal ciliary epithelium stopped short at the edge of this staphylomatous area, but the actual growth had extended beyond the area of the staphyloma round the entire circumference of the latter, overlying the surface of, but not invading, the normal epithelium. This extension

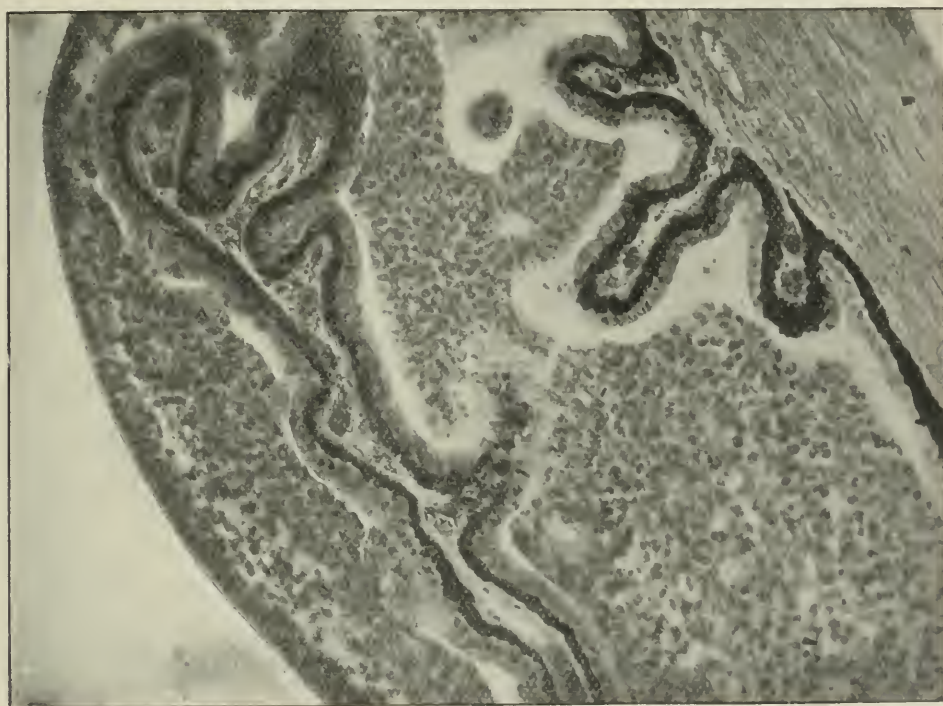


FIG. 3.

Section through main mass of growth, showing the ciliary processes embedded in the growth, but not invaded.

of the growth was most marked posteriorly, where the ciliary processes were overlain and surmounted by the growth in a most striking way, without any trace of invasion (fig 3).

The new growth was represented in the eye by three different kinds of structure: (1) At the place of origin of the growth a small solid mass, continuous with which were (2) cellular membranes which lined the lens capsule and branched among the anterior fibres of the vitreous body; (3) numerous cystic bodies, of which some were attached to the

cellular membranes, and some floated free in the anterior chamber and vitreous. It will be best to deal with these separately:—

(1) The solid mass (figs. 3 and 4) was situated just behind the posterior margin of the ciliary staphyloma and, as has been already described, surrounded but did not invade the ciliary processes in its neighbourhood. Its free surface was covered by a membrane, beneath which was a closely packed layer of nuclei showing the same arrangement as the outer layer of nuclei in the cyst walls. The main mass of the growth consisted of round nuclei similar to those which are found

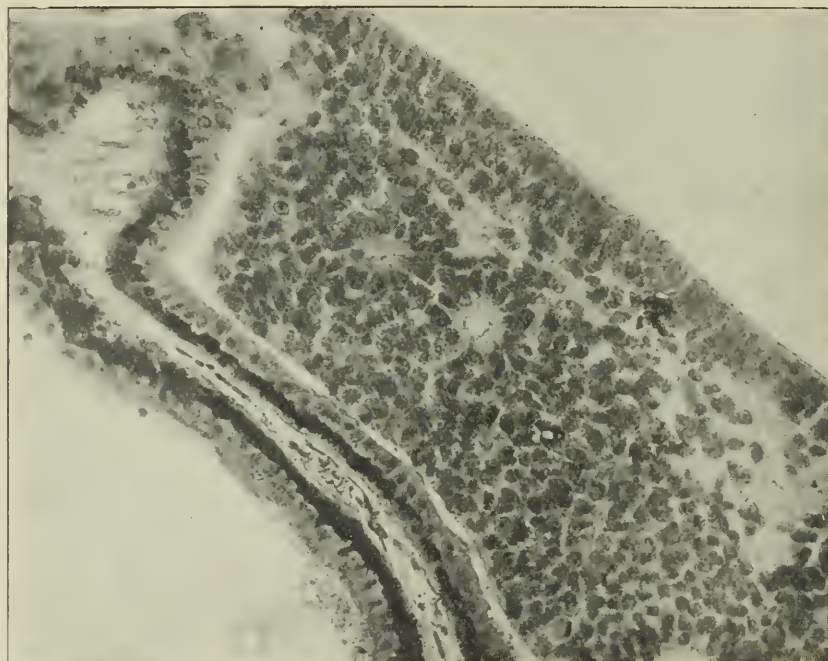


FIG. 4.

Section through main mass of growth, showing a well-marked rosette.

in a glioma of the pars optica retinae. They did not, however, take the hæmatoxylin stain so deeply as glioma nuclei. Rosettes of various sizes were abundant (fig. 4) of which the smallest sizes were identical in appearance with the rosettes of a true glioma (fig. 5). The thinned sclera which forms the wall of the staphyloma was not only eroded but also invaded by the growth. Solid columns of epithelial cells were present among the scleral fibres, and in one place a cell column of this nature could be seen to have invaded the wall of a blood-vessel (fig. 11).

(2) The cell membranes appeared in section as cellular strands, each consisting of a single layer of cells. These membranes clothed the lens

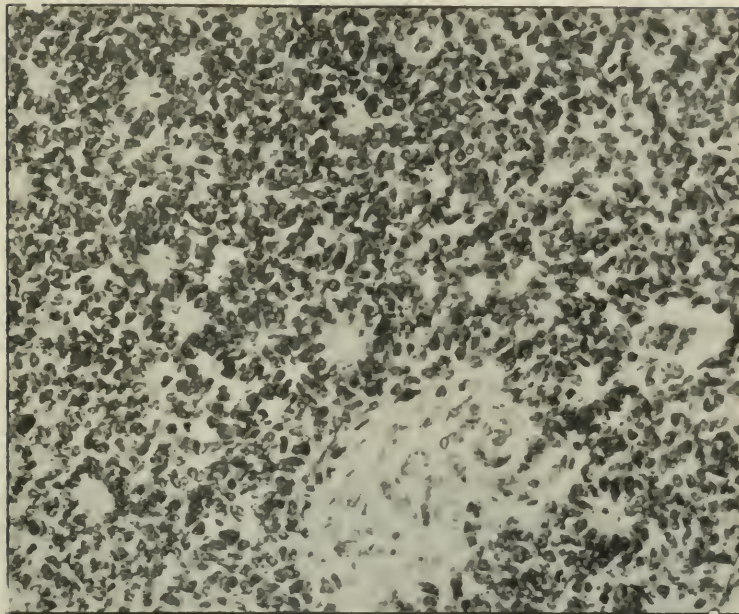


FIG. 5.

Section of a glioma of the pars optica retinae (from another case).



FIG. 6.

Section through lower half of anterior part of eye, showing free cysts in anterior chamber; also showing epithelial membranes clothing the iris.

capsule and the anterior fibrils of the vitreous, spreading downwards and to some extent backwards. They formed a complicated system of convolutions and tube-like formations which ramified in the posterior chamber and anterior part of the vitreous. A cellular membrane of this nature (fig. 6) lined the posterior surface of the iris in its lower and outer quadrants and passed round the pupillary margin so as to clothe the central part of the anterior surface as well. The cells which constituted these membranes varied a good deal in size and shape, being in some places tall and columnar, in others cubical, and again in others

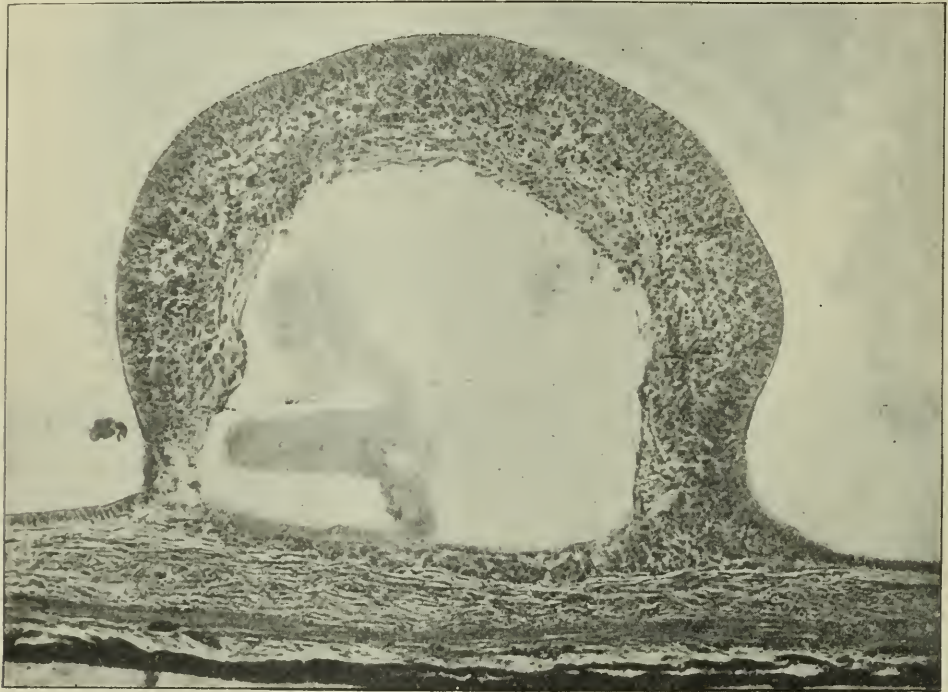


FIG. 7.

Section through iris, showing cyst arising from its anterior surface. Pigment epithelium seen below.

flat—the shape probably depended on the supply of nutrition. The membrane in certain spots became altered into denser cell masses which apparently had taken on the power of forming cysts. Several of these cyst-forming areas were present on the anterior surface of the iris, and some of them were budding out into single cysts (fig. 7), while others were giving rise to several cysts at once (fig. 6). The cysts in the vitreous were apparently formed by backward prolongations of these cell membranes.

(3) The cysts proper were both fixed and free and apparently were able to arise from any part of the growth or cell membranes (fig. 12). The fully formed cyst wall has already been described above (fig. 1); but though this structure could be seen in places, in most of the cysts the character of the walls varied a good deal. In some places the wall was reduced to a single row of cells, and in others, especially in the larger cysts, large necrotic areas appeared. The presence of necrosis is not surprising, owing to the complete absence of any vascular supply in any part of the growth, including the parent mass. The presence of these necrotic areas in the cyst walls probably accounts for the

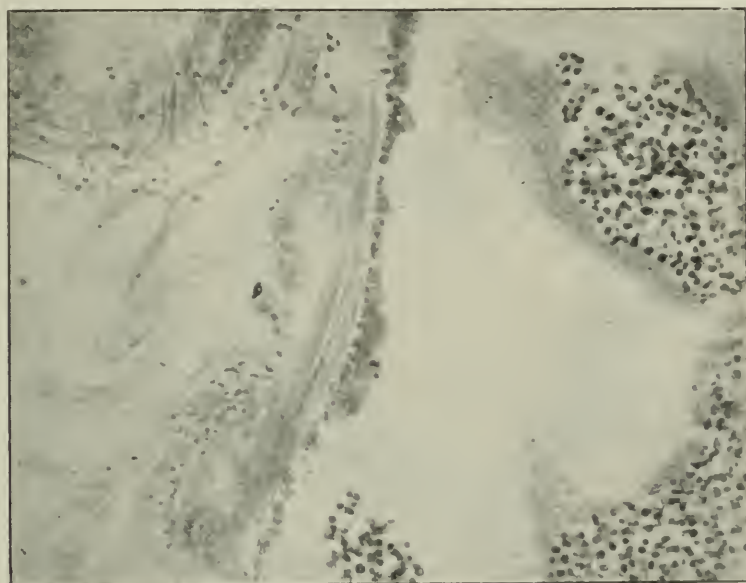


FIG. 8.

Section showing the contents of a cyst, consisting of vitreous-like substance and degenerated nuclei.

disappearance of the cysts when they reached a certain size. That such disappearances took place is undoubted, owing to the clinical observation that at the time of enucleation the free cysts in the anterior chamber were less numerous than they had been a week earlier.

The contents of the cysts were of a very interesting and remarkable nature. Many of the cysts, both fixed and free, contained a fibrillar substance, very closely resembling the normal vitreous body in structure. The fibrils of this substance occurred in long strands (fig. 8), which in the youngest cysts had a coiled-up appearance, but in the older ones were

irregularly arranged. They did not in the least resemble the pseudo-fibrils of a coagulum, which invariably show a branched, tree-like appearance. These vitreous-like contents filled the cavity of the young cysts, but in many of the larger ones, which usually were rather pear-shaped, they were present only in the narrow part, the wider part being partially filled with necrotic cells and sometimes true coagulum; apparently the wider part had bulged out at a later date. Rosettes occurred in the thick parts of the walls of the larger cysts. Near the place of origin of the growth, in the region of the ciliary staphyloma,

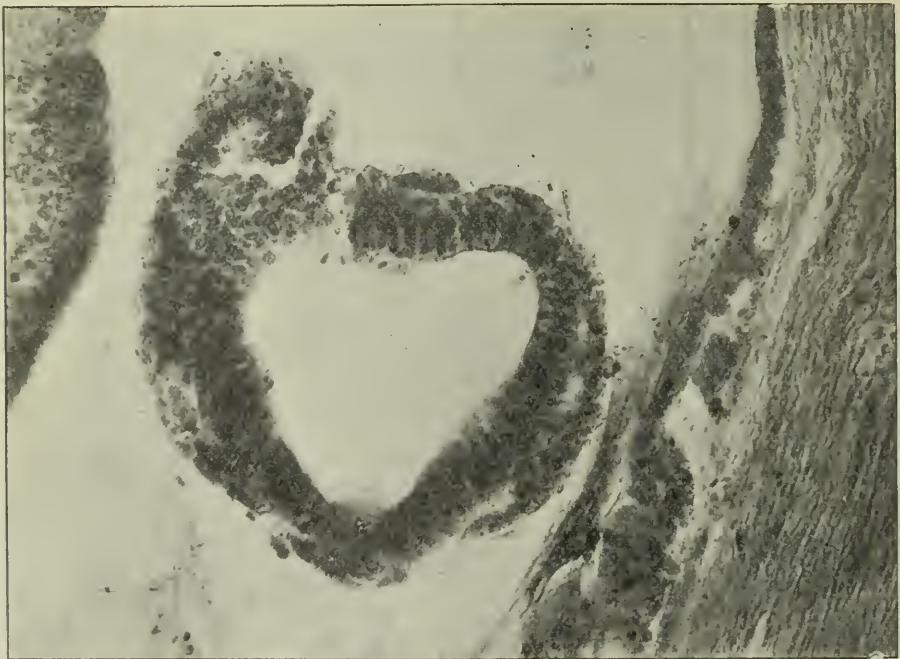


FIG. 9.

Section through a cyst, showing an inverted arrangement of the layers.

there were some small cysts, not numerous, the walls of which showed an opposite arrangement of the structures composing them to that found in the majority of the cysts. These walls, in fact, appeared to be turned inside out (fig. 9), so that the limiting membrane was found lining the lumen instead of encircling the outside of the cyst. The arrangement of the structures was exactly the same as in the other cysts, only in the reverse order from without inwards. A striking fact is that these particular cysts never showed any vitreous-like contents; they rather resembled very much enlarged rosettes. In a few places cysts could be seen budding off from other larger cysts (fig. 10).



FIG. 10.

Showing a small cyst budding off from a larger one.



FIG. 11.

Section showing the invasion of the sclera by growth cells. To the left a column of cells is seen to be invading a blood-vessel.

I will now describe briefly the condition of other parts of the eye. The cornea showed no abnormality. The iris was atrophic and its pupillary margin below was reduced to a mere strand of pigment epithelium, which was in places incorporated with the remains of the lens capsule and with some of the cellular membranes of the growth described already. These membranes had eroded and destroyed the lower part of the lens capsule, the upper part of which was, however, preserved and contained between its layers a small quantity of cataractous lens substance. Cellular membranes were found lining both the

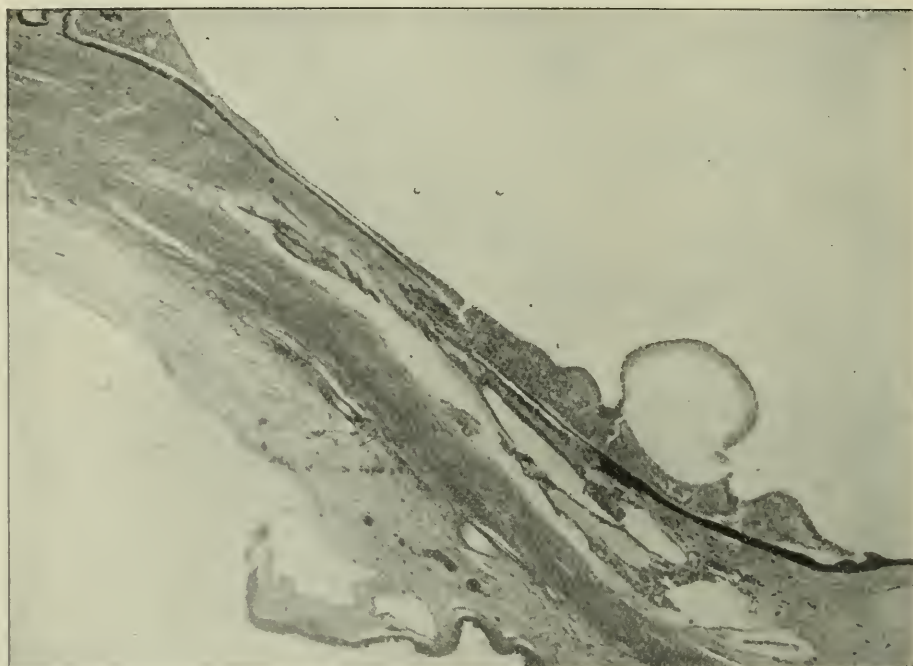


FIG. 12.

Section showing a cyst arising from the parent mass of growth.

anterior and posterior surfaces of the capsule. The angle of the anterior chamber was completely closed by a wide adhesion of the iris root, and a vascularised endothelial membrane clothed the entire anterior surface of the iris: this membrane was covered in the regions already mentioned by epithelial cells belonging to the new growth. Where the pupillary margin was free there was marked ectropion uveæ. The ciliary processes were fully developed, and the pars optica retinae was developmentally normal except for the presence of a number of small depressions in the equatorial region, in which all the layers of the retina were represented, but appeared to have been pressed together.

The ganglion and nerve-fibre layers of the retina were atrophied, as a result of the glaucomatous cupping of the papilla. No signs of active inflammation were seen anywhere—an occasional polymorphonuclear leucocyte was met with in the cysts, but very infrequently. No leucocytic deposits were found on the back of the cornea. The deposits which were observed clinically immediately before the eye was enucleated were probably debris from ruptured cysts. The choroid was normal and the optic nerve highly atrophic.

This case is, as far as I know, unique, clinically. As regards its main pathological characteristics, it is, however, the fifth of its kind on record. Fuchs, in 1908, collected particulars of all the cases of tumours which had been reported arising from the epithelium of the ciliary body. He was fortunate in being able to obtain and examine microscopic preparations of most of the growths, and he classified them into definite groups. With the so-called innocent growths of the ciliary body we are not concerned, and there is good reason for thinking that many of them were not new growths at all, but epithelial hyperplasias, secondary to senile or inflammatory changes in the respective eyes.

The malignant tumours were classified by Fuchs into two groups:—

(1) Those which imitate the structure of embryonic retina, showing several layers of cells in the membranes of the growth.

(2) Those which never show more than a single layer of cells in a row.

The second group, to which the present case obviously does not belong, is now represented by five cases, one of which—a case of unpigmented growth—was reported by me since Fuchs's paper appeared. The growths of this group may arise from either layer of the ciliary epithelium, and hence may be pigmented or unpigmented. They may occur in children or adults, and invariably show definite malignant characteristics.

The first group, to which the present case undoubtedly belongs, and which includes only growths arising from the unpigmented ciliary epithelium, is represented by four cases in addition to the present one. These cases are almost identical as regards pathological appearances, and show certain clinical points of similarity. Thus, all the patients were children under 9 years of age, and in each case the eye became glaucomatous and developed a ciliary staphyloma. In each case, too, there was a commencing invasion of the sclera, but the rate of growth was invariably slow and no other evidence of malignancy was present.

It is only because of the scleral invasion that Fuchs classifies them among the malignant tumours.

The present case shows two outstanding differences from all the rest:—

(1) Multiple fully formed cysts became severed from the parent growth, and appeared clinically as free bodies in the anterior chamber.

(2) While blood-vessels, though scanty, were present in the growth in all the other cases, they were absent from all parts of the growth in the present case. In this particular the growth differs markedly from a glioma of the pars optica retinae.

One curious clinical feature of the present case is the circumstance that the growth was seen first in the lower part of the eye, although there can be no doubt that its place of origin was above. The only explanation possible is that the part which appeared first was a continuation of the growth which had spread downwards from above and was then sprouting from the lower margin of the iris.

It is particularly difficult to find an appropriate name which shall express in one word the nature of this unique type of new growth. Each individual who has reported a case has suggested a different name, but none of these names are entirely satisfactory. It seems impossible to call the growth anything but an embryonic retinal new growth of the unpigmented ciliary epithelium.

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- FUCHS. *Archiv f. Ophthalm.*, Leipz., 1908, lxxviii, p. 534. (Fuchs gives references to other cases.)
 GREEVES. *Trans. Ophthalm. Soc. U.K.*, 1911, xxxi, p. 261.
 VERHOEFF. *Trans. Amer. Ophthalm. Soc.*, Hartford, U.S.A., 1904, x, p. 351.

DISCUSSION.

Mr. TREACHER COLLINS said he considered that one of the most interesting points in this very interesting case, was the way in which it demonstrated how pathology helped to explain embryological processes. It used to be generally thought that the vitreous humour was of mesoblastic origin; but of late embryologists had regarded it as derived from neural epiblast; and this specimen bore out that view very conclusively. Further, it showed not only that the vitreous humour was derived from the secondary optic vesicle, but that it came from only the front part. Mr. Greeves had demonstrated that vitreous humour was present in these cysts derived from the pars ciliaris retinae. In sections of gliomata of pars optica retinae a new formation of vitreous tissue was never found. It appeared, therefore, that vitreous humour was formed by the pars ciliaris retinae. He had had an opportunity of microscopically examining the sections of this specimen, and he thought they showed in some of the cysts not only vitreous humour, but also embryonic fibres of the suspensory ligament; at any rate, they showed branching cells very similar to what he had found in the circumlental space in microphthalmic eyes, where the fibres of the suspensory ligament were imperfectly developed.

Mr. MAYOU said he had not had such a case, but he would have liked to hear Mr. Greeves make some remarks about unpigmented tumours of the pars ciliaris. The point that gentleman raised was, that these were mostly epithelial hyperplasias in old people. A case he had was in a microphthalmic eye, and was of the pigmented type, very different from the type of tumour Mr. Greeves had been referring to. There was no evidence of malignancy. The eye removed was microphthalmic and behind the lens there was a mass of an unknown nature. The microscopic examination showed it to be the retina which had never come in contact with the outer pigmented layer of the retina, and between the angle so formed there was a pigmented tumour, composed of epithelial cells. It occupied about one-eighth the segments of the ciliary body.

Mr. W. LANG said it was many years since Dr. Barrett, of Melbourne, and he went to Liège to see Professor Fuchs, who was then the Professor of Ophthalmology in that University. One of the cases he showed them was a solitary cyst in the anterior chamber, which he had been watching for many years; and there appeared to be nothing else amiss with the eye. It was a semi-translucent brownish cyst. To the Professor at that time it was a mystery where it originated, but he was inclined to think it was a cell off the front of the iris, which had got shut off and was free. Since then he (the speaker) had had a similar case of his own. He tried to get it out by opening the anterior chamber, but it did not succeed, and the eye did badly afterwards: some chronic inflammation ensued, and eventually it came to excision. There was nothing to indicate where the cyst came from, or what was its cause.

Mr. GREEVES, in reply, said there were recorded about ten cases of single free cyst in the anterior chamber. He had seen a case similar to the one just mentioned by Mr. Lang; it was shown by Mr. Coats at a meeting of the Ophthalmological Society about two years ago.¹ There was no satisfactory pathological report of any of these cases, because, apparently, the cysts had not caused trouble, and so there was no justification for their removal. In all the cases the cysts had been pigmented and single. They were of long duration, and while most of them had been watched for some time, without change, others had undergone alterations of various kinds. Their nature was obviously different to that of the cysts in the case just described.

Mr. A. H. P. DAWNAY exhibited a "Case of Metastatic Carcinoma," and Mr. N. BISHOP HARMAN a "Case of Congenital Entropion."

¹ *Trans. Ophth. Soc. U.K.*, 1912, xxxii, p. 189.

Section of Ophthalmology.

December 2, 1914.

MR. PRIESTLEY SMITH, F.R.C.S., President of the Section, in the Chair.

Small Optical Iridectomies in a Case of Lamellar Cataract.

By Sir ANDERSON CRITCHETT, Bt., C.V.O.

SIR ANDERSON CRITCHETT said the condition in this child (A. S., aged 9 months) seemed so ideal for the operation he performed, that he decided to do these little iridectomies. He knew that, especially on the continent, the fiat had gone forth that, given a lamellar cataract, the lens should always be broken up. His father initiated this operation fifty years ago, and he (the speaker) later on assisted him, cutting off the little piece of iris. He read a paper on it at the Worcester meeting (British Medical Association, 1882), and performed the operation at St. Mary's Hospital before members of the International Medical Congress in 1881. The case suited for the operation was one in which there was a small, well-defined nucleus and an absolutely clear margin. The operation was done under an anæsthetic, with a small needle bent at an angle; the instrument should be very sharp. Having selected the exact spot of entry, the operator should plunge boldly; hesitation might cause him to run in between the layers of the cornea, and then it would be difficult for him to get his small hook in, and still more difficult to withdraw it. One great advantage was that the patient was well in about twenty-four hours, as the reaction was practically *nil*. The objection raised was that eccentric vision only resulted. That was true, but in such a case as this it was slight. He had had cases with J.1, and $\frac{6}{1\frac{1}{2}}$, and in one a few letters of $\frac{6}{3}$. There was always a risk, even in children, in breaking up a lamellar cataract, repeated anæsthetics were necessary, and matters did not always go as well as one would wish. But in this operation one had not burned one's boats, because, if necessary, it could be followed by discission. With regard to increase of cataract in the margin, he saw one case last year in which

his father had done the operation more than forty years ago and the margin of each lens was still absolutely clear. He hoped that, in quite suitable cases, this operation would continue to be regarded as a valuable asset in operative ophthalmology.

DISCUSSION.

Mr. W. H. H. JESSOP said he was very glad to hear Sir Anderson Critchett bring this operation forward, because it was one he had himself performed with great success, in adults as well as in children. He was reminded of a man twenty-two years ago upon whom he operated when 20 years of age. He had been brought up practically as a blind boy and could only read J.19; the lamellar cataracts were large. On dilating the pupils the vision was much improved, and a double iridectomy was done downwards and inwards in each eye. Ever since the operation his vision had been $\frac{6}{12}$ in one eye and $\frac{6}{9}$ in the other. Since the operation he had climbed several of the smaller snow peaks in Switzerland, and read practically every day. The margins of the lenses were still quite clear.

Dr. ANGUS MACGILLIVRAY (Dundee) expressed his indebtedness to Sir Anderson Critchett for bringing this case forward. In lamellar cataracts when the opacity was small, dense and sharply defined, optical iridectomy gave as a rule better results than extraction. The results following the removal of the lens were not so satisfactory in lamellar as in other cataracts. He agreed that the opacity in lamellar cataract did not spread provided there were no riders present, and he had watched cases for nearly a quarter of a century.

Case of Mikulicz's Disease.

By LESLIE PATON, F.R.C.S.

MRS. S. B., aged 62, came to St. Mary's Hospital on July 31, on account of the pain in her eyelids and eyes, and the swellings in her eyelids.

History: About two years before she had noticed some small lumps in the upper part of the back of the neck. These gradually reached the size of a large pea without causing pain or inconvenience. A year later lumps began to appear under the lower jaw on both sides (right shortly before left), and in the upper lids. Those in the upper lids are the only ones which have caused any pain or inconvenience.

On examination, there were seen to be large symmetrical swellings in the position of the lachrymal glands, and obviously involving both

the orbital and the palpebral portions of the glands on both sides. The eyes were displaced downwards and inwards, and so forcibly that she could not get her lower lid up to cover the eyes. The eyeballs were pressed against the lower inner margin of the orbit. The corneæ were both desiccated in the lower part, which accounted for a good deal of the discomfort. The swelling of the submaxillary glands gave rise to two large sausage-like rolls under the lower edges of the



Case of Mikulicz's disease.

lower jaw. This was so marked as to obscure the swelling of the parotid glands, which could, however, be felt projecting forward on the surface of the jaw in front of both ears. Behind the main swellings in the neck were several small, hard, swollen lymphatic glands, and also one or two in the posterior triangles. On opening the mouth, the palatal glands were markedly swollen, so that there were two elliptical swellings, with a deep groove between them, with a small ulcer on the right side and a larger ulcer on the left side.

She was admitted to the hospital under the care of Dr. Harris, who found that there were a few lumps palpable in the abdomen in the right lumbar region and two in the umbilical region. A blood examination was made: Total leucocytes, 20,500; small lymphocytes, 76.5 per cent.; large lymphocytes, 12.5 per cent.; polymorphonuclears, 11 per cent. Many disintegrated white cells in film. Red cells normal size and shape, but appeared vacuolated, and took on rather a bluish tinge with Leishman's stain. Wassermann reaction negative; urine, specific gravity 1015, acid, no albumin nor sugar.

A further blood examination made a few days ago shows an increase in the number of leucocytes, but the proportion remains much the same.

While in the hospital her temperature remained steadily normal and her general health good. The swellings seemed to decrease in size under treatment with arsenic. Since she went out they have again increased in size. The sublinguals did not seem to be affected, nor the glands at the sides and tip of the tongue.

I have shown this as a case of Mikulicz's disease, as I think that in its main features it agrees very closely with the description originally given by v. Mikulicz [7] in Billroth's "Festschrift" in 1892. The predominant feature is a symmetrical enlargement of serous glands about the head and neck, including the lachrymal glands, the submaxillary and parotid glands, the sublingual (not in the present case), and the glands in the palate and at the tip of the tongue. This enlargement was shown by Kümmel [6] to be due to a growth of the nature of a lymphoma in the interstitial tissue of the glands with a loss of true glandular structure. In the earlier cases there were no blood changes described, but von Brunn [1] in 1905 divided the cases into two categories: (a) without, and (b) with blood changes.

The disease is obviously related on the one hand to Hodgkin's disease, or pseudo-leukæmia, in which lymphatic glands are involved without there being necessarily blood changes; and on the other hand to leukæmia of the lymphatic type. In the present case the blood changes were definitely of that kind, though, considering the aggravated character of the disease, the blood was not profoundly altered. I rather think that the blood change in the present case is entirely secondary to the gland change. There are established large factories of small lymphocytes entirely replacing all these serous glands, and these must necessarily have, after they reach a certain stage of development, a modifying effect on the blood-stream.

I have seen no account of any English case, but Elliot [2] gives an excellent account of a typical case he saw in India.

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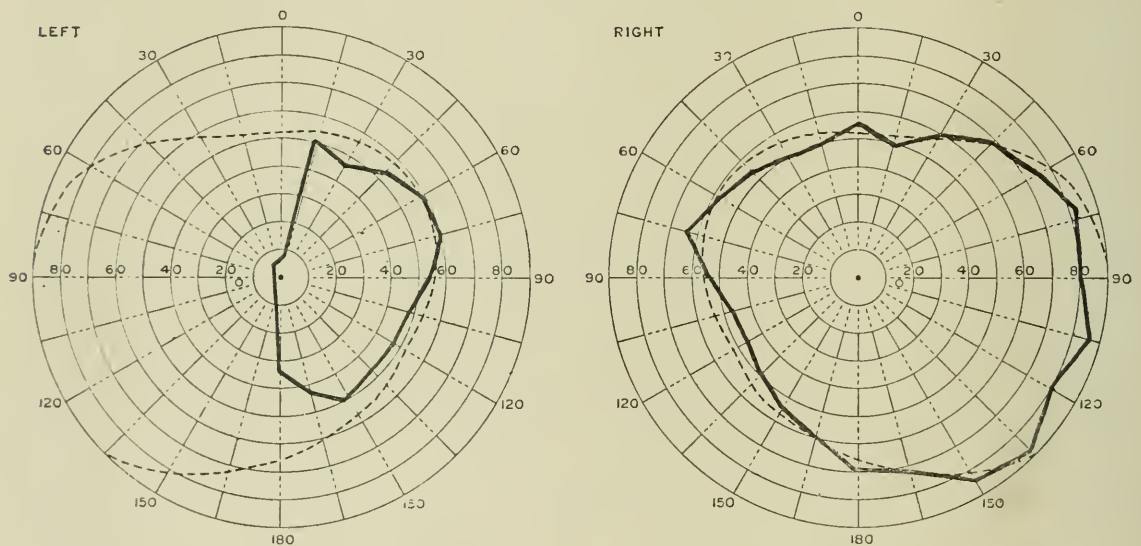
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Lieut-Colonel R. H. ELLIOT: In the *Ophthalmoscope* for February, 1911, I published notes and photographs of a case of Mikulicz's disease in a Hindu woman, aged 49. The lachrymal glands on both sides bulged like short horns, and the accessory portions of these glands projected into the conjunctival sac, like almonds let in under the membrane. The drooping of the lids gave the appearance described by Ziegler as that of "the blood-hound eye." The parotid, submaxillary and sublingual glands shared in the enlargement, but all the other glands were free. Medicinal treatment failed, and the patient demanded operation. The larger right lachrymal tumour was accordingly removed. It measured 47 mm. antero-posteriorly and 35 mm. laterally. Three weeks later the left growth was removed, but in doing so the conjunctival sac was entered at one point where the tumour was adherent to the membrane. The wound became infected, evidently through this opening, but the infection was easily controlled. The point of interest is that during this infection the corresponding parotid, sublingual and submaxillary glands showed distinct fullness and felt harder than before. This observation is to be taken with the patient's statement that the disease began in the lachrymal glands following an attack of conjunctivitis, which is well known to be a feature of many cases of this disease. The removal of the lachrymal glands not only satisfied the woman's desire for an improvement in her appearance, but exercised a distinct influence on the other glands affected. Captain Ingram furnished a pathological report on the case, which appears *in extenso* in the *Ophthalmoscope*. The leading features of it are (1) that the growth was more advanced in the lachrymal glands than in the parotid and submaxillary glands, portions of which were removed for comparison, but otherwise the structure was identical in all the glands; (2) the tissues examined presented a close resemblance to that found in round-celled sarcomata; (3) evidence of inflammation was absent. Ingram summed up the growth as a "multiple cellular growth affecting a number of glands," and of such a nature that the term "lymphomatous" was perhaps the most suitable to apply to it.

Case of Bilateral Temporary Hemianopia; Rapid and Permanent Recovery of Vision after the Administration of Thyroid Extract.

By H. L. EASON, M.S.

AT the combined meeting of the Sections of Neurology and Ophthalmology of this Society in March, 1913,¹ I produced notes of three cases of probable tumour of the pituitary body. The first of



Perimeter charts illustrating case of bilateral temporary hemianopia.

these had come under my care in the first instance in 1906; at the time of his admission into Guy's Hospital at that date he was practically blind, but recovered normal vision and nearly a full field in the right eye after only a month's treatment with extract of thyroid gland. Five years afterwards his vision was unaltered, but at the time of the meeting the patient was in South America and could not be exhibited. He has recently returned, and I am therefore taking the opportunity of showing him. The vision in the right eye is still $\frac{6}{6}$ and in the left eye $\frac{6}{60}$. The fields of vision are shown in the charts appended. Both disks are white, and a skiagram taken yesterday shows that the sella turcica, though little enlarged, is unusual in shape;

¹ *Proceedings*, 1913, vi (Sect. Neur. and Opth.), pp. i-viii.

PLATE I.



Angioid streaks.

*SPICER: Angioid Streaks in Brother and Sister; a suggestion
that the Streaks are Non-vascular.*

it is deeper than usual, and the anterior and posterior clinoid processes do not overhang in the normal manner. It is now nine years since he recovered his vision, but the patient finds that unless he takes small doses of thyroid gland from time to time his vision begins to fail and he suffers from headaches.

Angeioid Streaks in Brother and Sister ; a suggestion that the Streaks are Non-vascular.

By W. T. HOLMES SPICER, F.R.C.S.

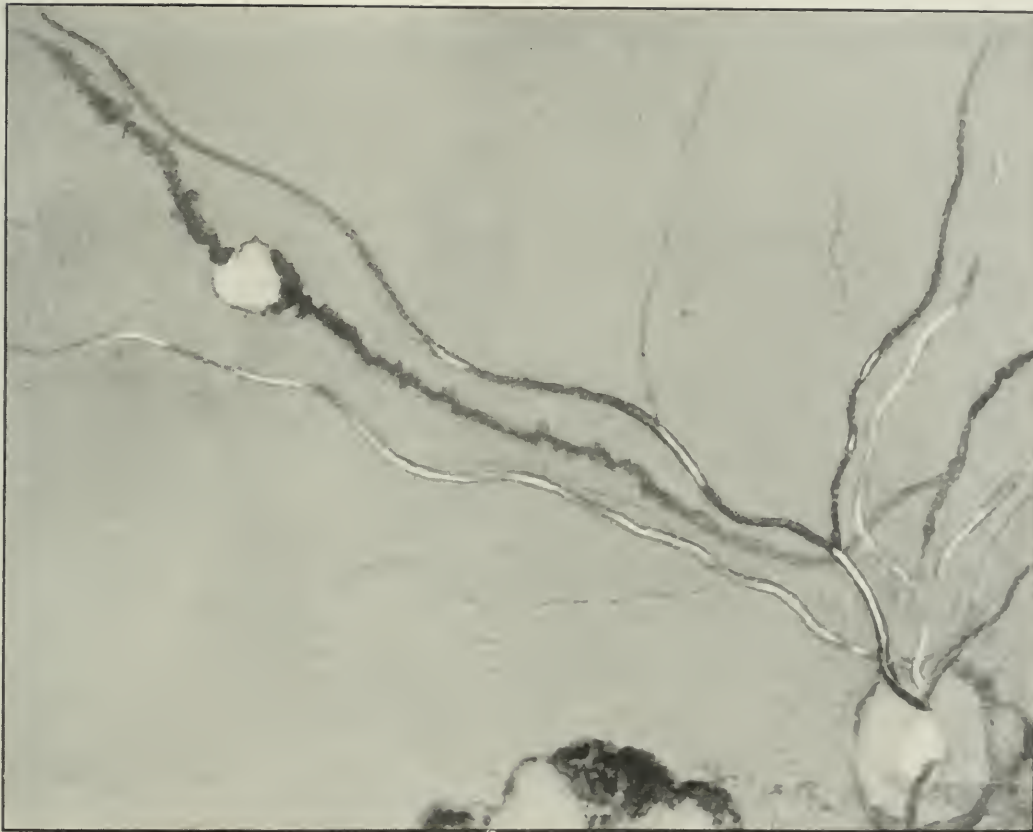
F. R., FEMALE, aged 38, was first seen at Moorfields on February 25, 1913. The sight began to fail three years before, just before her sixth child was born ; a gradual mist came over things. She does not think her sight was ever very good as she never learned to read well. The sight had got worse in the past month. She had fairly good health, had had seven children ; had one miscarriage, which would have been her fourth child ; her children were healthy. Vision : R., counts fingers : L., $\frac{6}{60}$. Both disks are rather pale, and the retina radially striated round the disk. There are fine pigmentary changes all over the central region ; at the yellow spot there is a good deal of new deposit of pigment and of fibrous tissue, which seems to lie on the surface of the retina. In both eyes there are conspicuous angeioid streaks, which appear to start from a circle round the disk, seen rather faintly, and resembling the edge of a myopic crescent ; the circle is not complete, the yellow spot side being free. From the outer edge of this pigmentary circle a number of irregular lines of pigment radiate out towards the periphery of the fundus ; they lie beneath the retinal vessels and increase in size and definition as they pass outwards. They are not perfectly black ; where they are thin they appear red-brown in colour, suggesting blood. They do not suggest retinal vessels near the disk, but they assume more and more the character of retinal veins as they pass outwards, both in definition and density. At the extreme outer periphery they break up into smaller branches which come to an end rather abruptly. The streaks are nowhere seen to communicate with any blood-vessels (Plate I). Her urine was normal. Heart sounds normal ; her pulse tension was so low as not to be measurable ; the radial arteries could scarcely be felt, but there was nothing in her appearance to suggest any constitutional disease.

F. B., male, aged 40, was first seen at Moorfields by Mr. Stanford Morton in 1907. He had found out, by covering the right eye, that the left eye was defective. As he had been in the Navy his sight in both eyes must have been good in earlier life. In May, 1907, his vision was: R., $\frac{6}{6}$; L., $\frac{1}{60}$. He had what was described as a large subhyaloid hæmorrhage at the yellow spot in the right eye, with a central patch of absorption where the choroid seemed to have disappeared, crossed by a small vessel. In the left eye he had a large patch of central choroidal degeneration, with great pigmentation at the edge and with absence of choroid at the centre. In both eyes there were angeioid streaks running very much the same course as the vessels of the retina, beginning faintly at the region of the disk, getting more distinct as they passed outwards towards the periphery, and more like retinal veins. In the right eye, in the course of the streak corresponding to the upper temporal vessels of the retina, the pigment column of the streak or vessel was interrupted by a small spot of complete choroidal atrophy; the streak reappeared beyond the atrophic spot (Plate II). If the streak were a blood-vessel it would be almost impossible for it to run into an atrophic spot in the choroid and reappear beyond it. It suggests very strongly that the streak is a pigment line only and not a blood-vessel.

This man and woman were brother and sister; they both presented the somewhat rare ophthalmoscopic condition of angeioid streaks. It was not a congenital peculiarity, the loss of sight had been recent in both patients; in each of them it had first appeared at about 35 years of age. The condition of both eyes of each patient was the same; they appeared to have an extensive macular degeneration which, in one eye at its first appearance, was described as a subhyaloid hæmorrhage; the peculiar lines of pigment stretched out from the centre to the periphery after the manner of retinal vessels. Both patients were in ordinary good health, with no disease of any of the important organs of the body, and with no appearance of congenital taint of any kind. Whatever might have been the cause, either their tissues had a similar want of power of resistance or else they reacted in a similar way to some poison, the nature of which is unknown.

Another point of interest in the case of the man lay in the fact that the streak was interrupted at one point by an area of choroidal atrophy, so that it is difficult to think that the streak is a blood-vessel, and goes to support the view that the streaks are lines of choroidal pigment only, and therefore are not angeioid.

PLATE II.



Angioid streak showing interruption in its course by an atrophic choroidal patch.

DISCUSSION ON THE NECESSITY FOR AN EXACT DEFINITION OF BLINDNESS.

The Exact Definition of Blindness.

By HAROLD GRIMSDALE, F.R.C.S.

THE importance of defining, if possible, the state of "blindness" has for long exercised the minds of all those who are concerned in the teaching and maintenance of the blind, and the recent awakening of public interest in the question has made it all the more urgent to clear away the doubts about the matter. There is a strong tendency in modern legislation, which is in many respects at least praiseworthy, to attempt to make up to the less fortunate for the inequalities of fortune, and if any class deserve our aid and help, it is surely those who, themselves deprived of the great aid of vision, have to work and to compete against sighted people.

The recent appointment of a Committee to discuss the whole subject of blindness has made the prospect of legislation more within the range of present politics; but it is a fact, and a somewhat curious one, that so far the Committee has made little attempt to decide who are the people for whose benefit it is sitting. The President of the Local Government Board invited me to become a member of this Committee, and as a result of this entirely unexpected honour I found myself there the sole representative of ophthalmic surgery. Therefore, feeling the importance of the occasion, and knowing my inability to answer for anyone but myself, I have ventured to bring the subject before the Ophthalmological Section of the Royal Society of Medicine, in the hope that members will recognise the necessity of action and the advisability of something approaching unanimity, and will give their help in answering the chief question which has arisen — i.e., the definition of who are blind, so that I may go back to the Committee and act in some degree as the spokesman of the Section.

It was with the hope of helping the Committee to decide, that recently I have sent out a circular letter, which many of you have seen, asking for the opinions of members on the point, and I should like to

take this opportunity of thanking all those who took the trouble to answer the questions which were there put down. I believe I have thanked each individually, but if any have not received thanks, may I ask them to accept them now.

The result of the circular letter has been to show that a large number of members of the Section consider it impossible to define the status of a blind child otherwise than is done in the Act of 1893—i.e., that for educational purposes a child shall be considered blind who is too blind to read the ordinary school books. Others say that no definition is possible, and that each case must be considered on its merits. This position is impossible already if we are to work in accordance with the Act, for in the first place the Act gives a definition of a blind child, and by contrast that of a seeing one, so that we must suppose that every child who is able to read the ordinary school books should by the Act be treated as fit for an ordinary school; yet there are many instances, which will occur to you at once, subjects of high myopia, &c., in which this is undesirable. Further, we cannot hope for a uniform inspiration to come to all of us, and we must therefore have some rules to guide us in the investigation and evaluation of each case. The rules at present are not rigid, but they must be there. If we can lay these rules down for a sufficient number of individual cases, we shall at least get some general idea as to the treatment of the problem, and on these ideas as foundation we may build later. Therefore, again, I ask you to give this matter your close attention.

The difficulty of definition is obvious. There are so many factors to be taken into account with regard both to the state of the patient and to his employment. It seems clear that both central acuity and the state of the peripheral vision must be considered; the relative vision of the two eyes is another important factor; the presence or absence of binocular vision, and other points which will readily suggest themselves.

As regards the calling of the patient, we know that for certain employments a much greater acuity is needed than for others. Thus for most trades, as Berry pointed out, an acuity of $\frac{6}{12}$ is probably as good as $\frac{6}{6}$, but for certain handicrafts—e.g., watchmaking—not only is $\frac{6}{6}$ an advantage, but it is practically necessary to have a fair range of accommodation, so that simple presbyopia is a marked handicap; and, as a matter of fact, I am told by the working craft that few watchmakers go on beyond the age of 50 on this account.

Now if we accept one definition of blindness which has been proposed

in certain recent legislation, that "the expression 'blind' means too blind in the opinion of the local authority to perform work for which eyesight is ordinarily required," it may be that, taking the watchmaker's statement as correct, we shall be obliged to accept as blind an artificer of the age of 55 who has an acuity of $\frac{6}{6}$, but has presbyopia.

There is one definition which is easy; total blindness can be defined only as the state in which the person has no perception of light, but such persons form only a small proportion of those who are ranked among the blind, and amongst children the proportion is still smaller. Among adults the percentage becomes steadily greater, since the cases of blindness from atrophy of the optic nerve and glaucoma increase in rough proportion to the age. Optic atrophy and glaucoma are very rare in children. It is difficult to estimate the relative frequency of total and what may be called economic blindness, but it has been attempted on a small scale. Thus at one "blind" school, among sixty-eight children eight had no perception of light; and this proportion is, I think, rather high.

At the request of the Massachusetts Commission for the Blind, the Research Department of the Boston School for Social Workers made the following report (vide *Outlook for the Blind*, July, 1914): "A study of 2,021 eye patients treated during two years in the wards of three Boston hospitals shows that 0·6 per cent. were totally blind, and that 7·7 per cent. were 'practically blind'—i.e., had vision less than $\frac{6}{60}$ of normal—15·9 per cent. had seriously defective vision but could not be called blind." Of course, such statistics as these are obviously fallacious. The totally blind do not often seek relief; after one visit to the hospital they or their friends are convinced of the hopeless nature of the calamity which has befallen them, and they stay at home. On the other hand, those with seriously defective vision are to some extent buoyed up by hope, and if one hospital fails to aid they go from one clinic to another, and are recorded at each; thus the number quoted for such patients may well be too large.

The problem before the Committee of dealing with the blind of the country is not, in one sense, a large one, because the numbers of those affected are comparatively small. It is generally supposed that the total number in the United Kingdom is about 27,000, or at any rate not more than 30,000, but it is very difficult to estimate the numbers with any accuracy, for many reasons, and the chief of these is that there is no standard of blindness. The census of 1911 gave, or rather purported to give, the number of those totally blind; this number was 26,336.

It is certain that this cannot be accurate, or even approximately accurate. To show this I read quotations from the report on the census, but first I must premise that 1911 was the first year in which statistics of "total blindness" were asked for; previous censuses had apparently interested themselves with all those who are usually spoken of as blind.

"The Report on the Census of 1911 recommended that expert opinion should be taken as to what constitutes 'blindness,' but from the inquiries which we made prior to the Census of 1911 we found that there were many divergent opinions as to how blindness should be defined. A definition which had met with a certain amount of international acceptance, and the employment of which might therefore have in some degree conduced to international comparability of the returns, was rejected by the English experts consulted as inadequate to their requirements. With a view, therefore, to making the question as definite as possible in order to obtain uniformity of results, the schedule asked only for particulars of the 'totally blind,' but it is extremely doubtful whether the figures obtained accurately represent their numbers. The numbers so returned were 26,336, of whom 13,257 were males and 13,079 were females—that is, one person in every 1,370 is blind: one in 1,316 males, and one in 1,424 females."

As compared with previous censuses, there is a diminution in—

Year				Blind per million		Persons living to one blind
1851	...	18,306	...	1,021	...	979
1861	...	19,352	...	964	...	1,037
1871	...	21,590	...	951	...	1,052
1881	...	22,832	...	879	...	1,138
1891	...	23,467	...	809	...	1,236
1901	...	25,317	...	778	...	1,285
1911	...	26,336	...	730	...	1,370

Though there is a decrease in the percentage it is not so great as in some other decennia, and there can be little doubt that almost every one living who had been returned as blind in previous censuses was again returned as blind in this, though he was not totally blind. Thus, for example, in one institution with which I am acquainted all the inmates were placed in the census as totally blind, though probably not more than 10 per cent. of them are unable to perceive light. The report of the census says: "As regards the value of the figures for the — blind, however, it seems probable that a number of persons have been returned as totally blind who possess some degree of sight, for

it was apparent from discussion before the census with those interested in the welfare of the blind that the information desired in regard to this matter is rather that of the number of persons whose defect of vision is so serious as to forbid any ordinary means of earning a livelihood." The report continues that the compilers have found themselves unable to devise any means by which the ordinary mechanism of an English census could be adapted to discover the prevalence of economic blindness, but since to the layman economic blindness is of much more real interest and importance than total blindness, they think it probable that the public have been led to class many individuals as blind who still retain some amount of sight.

Nor are the figures available for computing the number of blind children more reliable. Dr. Eichholz gave in evidence before the Committee the statement that the number of totally blind children in England and Wales was 1,532, and the number of children blind within the meaning of the Act—i.e., incapacitated from reading school books—as 2,200. Now it is clear that these figures cannot both be accurate; for, as we have seen, the ratio of the totally blind to the economically blind is somewhere about as 1 to 9. If there are 1,532 totally blind children there must be some 15,000 who are economically blind; if, on the other hand, there are only 2,200 economically blind there will be about 250 totally blind, and this is more nearly correct.

Blindness is a handicap in two conditions or stages of development: (1) It makes education more difficult; and (2) prevents the sufferer from profiting fully from the education he has received. Legislation has to consider the problem of the blind from both these sides: (1) it has to arrange special measures to educate the blind; and (2) it has to consider and, if necessary, assist the wage-earning capacity of those who have been trained. And the two classes of people—those who are to be educated and those who must be assisted—are not necessarily the same, though to a large extent they must at least overlap. The blind child must be defined as one who cannot learn by the help of his eyes so easily as with his other senses. He may have sufficient sight to enable him to do work of many kinds in the world, but his sight is insufficient for reading the ordinary books which are the vehicle through which the normal child largely imbibes his instruction. Thus it does not follow that all children who are classed as blind for education purposes are going to form members of the class of adult blind; and, on the other hand, only a comparatively small part of the total blind population are blind from birth. Some three-quarters of the blind

lose their sight after school age, and, therefore, do not receive their training as blind people in the most assimilative age.

Thus there are three classes to be considered:—

(1) Those who are blind from childhood and whose vision is so defective that they must work at a “blind” trade.

(2) Those whose vision is so defective in childhood that they have to receive their education at a blind school and by “blind” training, but are able to work at some sighted trade when adult.

(3) Those who lose their sight after school age and who, therefore, receive their training as blind people later in life, at a time when they are less assimilative than the first two classes.

For purposes of education the first and second classes require largely the same treatment. The third class, which is by far the largest, is at once placed more favourably in some respects, and in others less favourably, than the first and second. It has the advantage of having received a sighted education, and therefore its education will probably have been (nay, rather certainly must have been) fuller than that of the first; for, consider how much of his education a child receives unconsciously through watching others. How much more copious is the literature open to the sighted. On the other hand, these sufferers are at the great disadvantage of being forced to begin a new training at a comparatively late age, when the mind assimilates new facts and methods more slowly; and if they are advanced beyond middle life their achievements in attaining a blind education are likely to be small.

The problem, then, divides itself into two parts: (1) What is a blind child? (2) What is a blind adult? The first half of the problem seems to be easier, and to offer more chance of a ready solution, for several reasons.

(1) We have already a definition of a blind child in the Education Act of 1893, that for purposes of education a child shall be considered blind who is too blind to read the ordinary school books; and it should not pass the wit of man to translate this into terms of distant visual acuity, as estimated by Snellen. If this can be done we give a much easier and less variable standard.

(2) The second point is that the requirements of children, as far as visual acuity is concerned, are more or less the same for all—i.e., reading school books, blackboard, &c., &c.; the range of work is little varied.

(3) Lastly, the causes of economic blindness in children are largely

different from those of adults. In children the chief trouble is loss of central acuity from corneal conditions, in adults the chief cause is disease of the optic nerve with contraction of the visual fields; in children the field is generally full, and when the blindness is due to disease of the optic nerve the state when it comes under our notice is not usually progressive.

We must consider one other thing; there is a tendency to make the standard of vision too high, and thus to throw a large number of children into the blind class. A child with bad sight demands more individual attention from his teachers, and is therefore to some extent a drag on the whole class. Both for his sake and for that of his fellows there is, then, an inducement to put him into a special school. This is cruel kindness to the child. It may for the moment ensure that his education is better looked after, but if a child is once labelled blind there is a risk that he may be drafted into a blind workshop after he leaves school, and thus a child with sight sufficient to enable him to earn his living in many trades may be impelled to enter one of the poorly paid trades which are open to "blind" people, and thus still further increase the difficulties of that trade.

The co-education of the blind and the seeing is one of the most interesting experiments that have been made by the schools of the United States. There the blind child is first started in a special school, until he has mastered the elements of Braille writing and reading, and then as soon as he is able to do so he takes his place among sighted children in the universal school, and competes, often successfully, against them. This is a most valuable training not only for the blind but also for the "blind seeing public," as Miss Holt calls them; for the mass of men have eyes but do not use them. The public see that blind children need not be and are not helpless, nor hopelessly handicapped by the loss of sight; the blind child learns self-reliance, and is brought out of his life of solitary introspection into the society of his fellows.

We have now to consider the definition which has already been mentioned—that of the Education Act of 1893—to see whether we can simplify it for our own purposes so as to make it more easily administered by the medical officers who have to decide on the status of the children.

"A child shall, for purposes of the Act, be considered blind who is unable to read the ordinary school books."

The ordinary school book is an uncertain term, but I think we may

take it that few school books are printed in smaller type than "small pica," J.6 or Snellen 1.2. Now the acuity required to read words of a given type is considerably less than that required to read isolated letters, since in reading we recognise the whole shape of the word and not that of each individual letter. It is difficult to get exact figures as to this, but the fact was brought to my notice a long while ago at the Physical Laboratories in Cambridge; there certain measurements were taken of a number of students, and it was a common thing for the examinee to be able to read J.1 at 30 in. or even 35 in. J.1 corresponds roughly to Snellen 0.3, and therefore this acuity approximates to three times normal. I have made a few experiments on this point, and find that the "near" acuity as measured by reading words is often more than twice the distant acuity measured on single letters. It follows from this that if the ordinary school book is taken at 1.2 Snellen, and the ordinary reading distance is 0.3 m., we have a large excess of acuity, and that a practised reader with even vision $\frac{6}{50}$ would probably be able to read 1.2 Snellen, at 0.3 m., with not very great fluency perhaps. With more than this amount of acuity there is little doubt that the child will learn more easily to read by means of his eyes than by his fingers; and with even less than this it will be found that so-called "blind" children try to read Braille by preference with their eyes instead of by touch; it is one of the difficulties of the blind school to prevent this use of their eyes by partially sighted children. But between the blind school and the ordinary school there should be an intermediate school for children who have such defective vision that they cannot be taught by the blackboard. However, this is not really a part of our immediate subject, and I therefore confine myself to suggesting that a distant acuity of $\frac{6}{60}$ should be taken as the highest limit of vision for a blind school child.

Among the answers which members have kindly sent there have been several which expressed the opinion that no elucidation of the definition of a blind child was possible or desirable. To these I would point out that we have a definition by which we are bound; as it stands, it implies that all children who can read the ordinary school books are fit to be educated in the ordinary schools. It takes no note of the distance at which the child reads; many children who are certainly fit for blind schools can read small pica after a fashion—holding the book against their noses. Many more—myopes—can read easily, but at a risk to themselves. It is true that Dr. Eichholz tells me that he reads the Act as meaning that the child shall be able to

read with no damage to himself, but this is not to be found in the Act, and is a gloss put in by his strong common-sense. I am not sure what the views of jurists would be on the point. Further, I would ask those who are satisfied with the definition, Do they test the children's vision with the ordinary schools books? Do they not rather themselves rely on the distant acuity?

To sum up shortly the conclusions to which I, personally, have come, may I say that I should be inclined to place no child in a blind school who has corrected vision more than $\frac{6}{60}$, but that I think there should be special schools for all children who have vision not more than $\frac{6}{24}$, and any others who are recommended for the special school, in which specially large type, special blackboards, &c., should be used, as in the myopic schools of the London County Council. All who have vision better than $\frac{6}{24}$, unless otherwise recommended by a medical man, should go to the ordinary school. Lastly, all the special schools should be inspected at least once a year and the vision of each child tested and recorded.

In adults the question obviously presents much greater difficulties; for, whereas in children we had already a legal definition of blindness, there is none here. The different societies and charities which look after the blind vary considerably in their acceptance of the term. Thus Hetherington's Charity for the Blind, a society which does much to relieve the blind by pensions, demands that they shall be totally blind, or, at most, able only to distinguish light from darkness. This definition includes several curable conditions, more particularly cataract, and excludes many which disable the sufferer absolutely from carrying on any useful employment by means of sight. It is also clear that a lax certificate will, or at least may, enable applicants who are really outside the limit to receive pensions, while a deserving applicant may be debarred by the conscientiousness of his medical man. We have seen reason to think that the census figures which are supposed to relate to total blindness are seriously at fault, and it is not unlikely that people who are willing to stretch a point for census purposes may also be willing to do so for securing a pension. Gardner's Trust for the Blind word their certificate, which must be given by a doctor, that "A. B. is for all practical purposes blind, and must be treated as a blind person." This is obviously the fair way to deal with the applicants, but it throws on the medical man the difficulty of deciding whether the applicant is for all purposes practically blind. It is, as a matter of fact, read somewhat loosely; we cannot really consider a

man who can guide himself readily by his eyes to be literally, for all purposes, practically blind, and yet many such are in receipt of help from the Trust. This definition has been adopted by many others of the charities.

It is clear that among the blind there will be always two classes—those who cannot see at all or have only light perception, and those who have sufficient sight to distinguish large objects, and are, therefore, able to guide themselves in unfamiliar surroundings by the aid of their eyes. An acuity of much below $\frac{3}{60}$ will enable a man to move freely in the outside world, if his field is good. We must all have met with agricultural labourers who, after cataract extraction, have preferred to go about without their correcting glasses. For this reason I regard the usually accepted $\frac{6}{60}$ as much too high to call practical blindness. I should feel inclined to put it at $\frac{3}{60}$, and then feel that I err on the side of leniency. For these two classes of blindness we should have names. An acquaintance of mine who is something of a “classic” has suggested that we might call the latter “autagogic” and the former “cynagogic,” meaning, I suppose, that the one can guide himself, while the latter has to rely on some extraneous aid, such as a dog. I am inclined to think that the word “cynagogic” would mean leading a dog rather than being led by one, and so it would be better to restrict its use to the malingerer who has a dog but does not need one; and in any case, I doubt if the words would meet with universal acceptance. Perhaps “totally blind” and “economically blind” will meet our needs.

There is the great difficulty of the different requirements of different trades—a point to which attention has already been drawn—and the varying adaptability of the individual. Some may be able to take up a new craft, if their vision has fallen to a point below the standard of economic blindness for their original trade; many, with less adaptability, may not.

Finally, there is the varying condition of the eye. In children, as we have said, blindness is chiefly due to disease other than of the optic nerve, and the condition is usually stationary, and not progressive, but in adults it is different. Disease of the optic nerve forms a large proportion of the causes of blindness, and is progressive; further, it brings in the varying acuity of the central and peripheral parts of the retina. Defects of the visual field may be as important to the adult as defects of the central acuity, in fact, it may be doubted whether a workman with Leber’s disease, a good field and a large central scotoma, vision $\frac{6}{60}$, is not more efficient than a man with central acuity of $\frac{6}{9}$ and a telescopic field of radius 5° .

In any scientific study of the relative efficiency of individuals the condition of the visual field must certainly be taken into account. Is it possible to do so practically, and readily in the question of a State definition of blindness? It seems to me that it is not possible except in the roughest way. And there is one additional factor which makes me think that it is not necessary to do so; this is the progressive nature of the diseases which occasion the contraction of the fields. Thus, though it would be a real hardship for a man with acuity of $\frac{6}{24}$ and a minute field to be denied a pension because of his high central acuity, it is not a permanent hardship in most cases, because, from the progressive nature of the diseases which give rise to these conditions, the central acuity will probably fall within a few months to such a point as will allow him to come within the range of definition, and it would then be possible for him to renew his application. There is bound to be hardship in some borderline cases in whatever way we decide to define blindness. If we accept some verbal definition, with no attempt at accuracy, such as that of the suggested Bill, which has been already quoted, "too blind, in the opinion of the local authority, to perform work for which eyesight is ordinarily required," we throw the onus of selecting cases on to the local authority, that is, as the Bill explains, the council of any county or county borough, a body who are not likely to be well qualified to judge on such a difficult point; for it must be remembered that the public are very ignorant on the question of blindness, even those who have to deal with the blind officially are not apparently aware of the degrees of blindness. Thus, I asked one of the official witnesses the question who were the blind in his district and how they were discovered. He replied, "Why, of course, the blind are those who can't see," in a tone which showed that he had no doubt about the matter. On the other hand, charitable feeling may carry local authority too far, as has happened (so Mr. Charles Campbell told me) in the United States of America. There there is a pension for every blind person—i.e., with visual acuity not greater than $\frac{6}{60}$ —at least in some States he (Mr. Campbell) had to investigate the claims of some of the pensioners. He found one who appeared to have very good vision, and asked the grounds for granting him the pension. He was told, "Well, he isn't exactly blind, but you see he has a wooden leg." Any such selection of candidates must be done by properly qualified medical men; no other body is fitted to examine and decide on the evidence of such cases. Nor is it fair to leave the individual medical man without some stated guide for his decision.

I give my own views on this point for what they are worth. I should accept any one with corrected vision of less than $\frac{3}{60}$ as *ipso facto* eligible for a blind pension. I should accept no one with vision above $\frac{6}{36}$, in any case. But between these two limits I should feel inclined to consider cases of defective vision with contracted fields or other abnormality. These should be adjudicated by a committee of experts, on lines laid down by this Section. For example, $\frac{6}{36}$ and a field of not more than 10° in any diameter, and $\frac{6}{60}$ with a field not exceeding 30° , might be accepted as the equivalent of $\frac{3}{60}$ with a good field.

Though this question is, as I have said before, a small one, if we consider only the number of the people affected, it is a very large one when we consider that it establishes the principle of making up to an inefficient worker for his inefficiency. It is a new principle that the State should pay to the inefficient worker the wages of an efficient one, or rather, perhaps, the difference between the two wages, and it is very important that the privilege should not be exercised except under the very closest supervision. If we allow that a man with bad sight, who works his best, should be paid, not for what he has done, but what the same care would have earned him if his sight had been normal, those who are incapacitated in other ways will claim the same privilege, and if we are not careful, a congenital disinclination to work may have to be paid for, since mental conditions are at least as real as physical.

In this latter part of the question the result of the circular letter has been to make clear the difficulty ahead of us, but in view of the real importance of the question I would suggest the appointment of a sub-committee to consider the whole matter in all its bearings.

Among the answers to this part of the question was one which puts so clearly the necessity for united action that I hope the writer will forgive my quoting it. "I fail to see how any question of partial blindness in relation to occupation can be settled simply. Only an ophthalmic surgeon can decide, and even he would require guidance if there was to be any uniformity in the definition of *blind* in relation to occupation."

Now it is this very guidance which I have hoped to help to obtain. The Bill to which I have referred would place the selection of the candidates for pensions in the hands of the local authorities, that is, as defined in the Bill, the council of any county or county borough. No mention is made of any examination by medical men, still less is there expressed any need for an examination by an ophthalmic surgeon. It is true that if the local authority has refused to consider an applicant as blind he

has the right to appeal to persons appointed by the Board of Education and a Secretary of State.

If we, as the leading authority on ophthalmology in the United Kingdom, refuse to consider the question seriously, we shall find a Bill passed through the Houses of Parliament without any reference to our opinion, and deservedly, and the most difficult questions will be left to the decision of absolutely unqualified amateurs.

Definition of Blindness.

By WILLIAM C. ROCKLIFFE, M.B.

MR. PRESIDENT AND GENTLEMEN,—As doubtless you are aware, there has been from time to time, in latter years, considerable agitation for State intervention on behalf of the blind, principally the outcome of a Socialistic society styling themselves “The National League for the Blind,” and composed almost entirely of the blind labouring class, led by the usual agitators.

In March this year Mr. Wardle (the Labour member for Stockport) moved a resolution “That in the opinion of this house, the present system of voluntary effort in aid of the blind people of the country does not adequately meet their necessities, and the State should make provision whereby capable blind people might be made industrially self-supporting and the incapable and infirm maintained in a proper and humane manner.”

Mr. Herbert Lewis (Secretary to the Local Government Board) added that the Government were prepared at the earliest possible moment to appoint an Inter-departmental Committee to inquire into the whole question, as an essential preliminary to any further State action.

In May this Committee, consisting of twelve, including Mr. H. Grimsdale, were appointed “to consider the present conditions of the blind in the United Kingdom and the means available” for (1) their industrial or professional training, (2) their assistance, and to make recommendations. Naturally they were at once faced with the question upon whom were they requested to adjudicate—i.e., who are the blind, or, in other words, “What is the definition of blindness?”

The necessity for such definition, I think Mr. Grimsdale’s paper most conclusively proves. For the last fifteen years, however, I have

been personally advocating some definition, and many letters have been published in the Press and elsewhere, which possibly some members present may have seen.

In 1902, at the Conference of the Blind, held in London, the Registrar-General quoted statistics, and I ventured to ask him for his "definition of blindness." He replied, "I suppose a man is blind when he cannot see." Again I had a similar answer from a supposed great authority on the blind in the North.

These two absurd replies stimulated me to further action, but nothing practical has so far resulted. I welcome, therefore, the possibility of some reliable definition being now arrived at.

Blindness has been defined, or was suggested by the present Departmental Committee, as (1) too blind in the opinion of the Local Board to perform work for which eyesight is ordinarily required; and by others as (2) blind for the practical purposes of life, (3) unable to follow their usual occupations, (4) guiding vision only, and (5) no perception of light. Clearly the first four definitions are useless from a scientific standpoint, and the latter is unfair to many.

It seems to me that to define the term "BLIND" (i.e., **those who should be eligible for admission into blind workshops, and to participate in the public contributions, pensions, and such-like given for the blind**) the opinion is required, not only of the medical profession, but also from those conversant with the life, occupation, and habits of the blind.

It so happens it has been my privilege for some thirty years to be not only Ophthalmic Surgeon, but also Hon. Secretary and Treasurer, and for many years Hon. Manager of our Hull Blind Institution, and, as such, being personally intimate with the homes and surroundings of all our blind, I humbly claim to have had some experience of those who ought to be classified as such. During this period I have examined and admitted some 800 blind on our books, and have certified several hundreds to our Corporation as eligible for free tram rides, and also a large number for pension societies. In every case I have used the following definition and no dispute has arisen. I divide the so-called blind of all ages into (1) blind, (2) partially blind or with defective vision; and include as *blind* (a) those with no perception of light, (b) those unable to differentiate, and (c) those able to differentiate, but at a less distance than 3 ft.; as *partially blind*, "those able to differentiate beyond 3 ft. but with less acuity of vision than $\frac{6}{60}$."

My reason for limiting the acuity of vision to fingers at 3 ft. in

a blind person is based on the practical knowledge that a blind workman who can differentiate beyond 3 ft. (or possibly even less) can distinguish his rods in basket-making, strands in cork fender making, bristles in brushes, canes in cane chair seating, more readily, and consequently works quicker and earns more, than his less fortunate fellow-workman, this resulting, where a bonus or augmentation of wages is distributed in proportion to wages earned (as in our Hull Blind Institution), in the more deserving receiving the lesser amount. This clearly should not be.

As before stated, I apply the above definition of blindness to all ages, but for the school education of the young I agree with Mr. Bishop Harman's expressed views—viz., children with less vision than $\frac{6}{60}$ should go to a blind school; children with more than $\frac{6}{60}$ and less than $\frac{6}{24}$ to a myope school; children with more than $\frac{6}{24}$ to an ordinary sighted school. I would therefore propose, if the above definition of blindness be approved by this Society, that Mr. Grimsdale submit the same to the Departmental Committee for their consideration and possible acceptance.

The Necessity for an Exact Definition of Blindness (Classification of the Blind).

By ANGUS MACGILLIVRAY, M.D.

FOR industrial or economic purposes so-called blind people are generally divided into three classes:—

Class I: Totally Blind.—Those who do not possess any perception of light.

Class II: Practically Blind.—Those who possess perception of light, but are unable to take part in any industrial occupation owing to their marked impairment of vision.

Class III: Partially Blind.—Those whose vision is such as to preclude them from obtaining employment in a sighted workshop, but whose vision is such as to disqualify them for admission to an institution for the blind on equal terms with those totally or practically blind.

The partially blind, coming between the sighted and the practically blind, are the most difficult to deal with from the economic

standpoint. They, on the one hand, are unable to compete with sighted people, and on the other, having a modicum of sight, they possess an obviously undue advantage over the totally or practically blind. In this class an intermediate industrial department is desiderated, where the work done is beyond the capacity of the totally and practically blind worker. The partially blind can with success engage in such work as cabinet making, joinery, &c., the finishing touches being done by a sighted workman. The presence, however, of a member of this class alongside of the totally or practically blind would not, as a general rule, be welcomed in a workshop where the work done is almost entirely piecework. In the institutions for the blind in this country some 30 per cent. of the inmates of the industrial department belong to the partially blind class.

From the educational standpoint so-called blind children can be divided into three classes similar to the adult blind:—

Class I: Totally Blind.—Those possessing no perception of light.

Class II: Practically Blind.—Those who possess perception of light, but whose vision is so impaired as to be useless for educational purposes.

Class III: Partially Blind.—Those whose vision is such that it is impracticable that they be taught alongside sighted children.

The totally and practically blind should be taught together in an ordinary blind school, but the partially blind, with advantage to themselves and others, had better be taught in an intermediate school where the educational methods are adapted to their special needs. To this intermediate school should be admitted such cases as progressive myopia with organic defects, or cases of any progressive condition of the eyes liable to be aggravated by the ordinary educational methods approved for normally sighted children.

Although the subdivision into the three classes just described for adults and children is that generally adopted by the management of the various institutions for the blind in Great Britain and Ireland, still no recognised standard of visual acuteness exists to guide medical officers as to the applicants' suitability for admission. Such tests as *seeing hand movements, counting and recognising fingers*, at varying distances are usually employed as the test of suitability, but these tests are obviously inaccurate. The only visual tests that can be regarded as accurate are those based on angular measurements as adopted by the late Professor Snellen. Instead of holding fingers in front of the applicant at varying distances, the large letter marked

D = 60, subtending an angle of five minutes, or the broken circle of Landolt, should be employed. The large test-types, black on white or vice versa, can, if properly lighted, be discerned more easily and accurately by applicants with very defective vision than fingers held in front of the examiner. By measuring the maximal distance in metres at which the letter marked D = 60, or the broken circle D = 60, can be seen, an exact measurement can be readily obtained.

Having found a quick and accurate method of determining the amount of visual acuteness in the practically and partially blind, it will now be necessary to determine what standard of vision is applicable to these classes. This can only be done by subjective tests similar to those adopted by Donders and Snellen when determining the smallest visual angle. The subdivision into these three classes described is generally recognised by the management of our institutions for the blind. If, with the co-operation of the manager, each ophthalmic surgeon of the sixty-six institutions for the blind in England (52), Scotland (7) and Ireland (7) were carefully to examine the visual acuteness of all the inmates in Classes II and III by the angular method advocated above, accurate records of each class would be available. The results of these examinations could be submitted to a Committee of the Ophthalmological Section of the Royal Society of Medicine, which would have no difficulty in drawing up the range of visual acuity applicable to each class. For example, if it were found that the visual acuteness of Class II, the practically blind, ranged from perception of light to $\frac{1}{60}$ of normal vision, then, in future, candidates possessing perception of light up to and including $\frac{1}{60}$, could with accuracy be placed in Class II, the practically blind. Those above $\frac{1}{60}$, but possessing vision unsuitable for economic purposes, could be placed in Class III, the partially blind. In the case of applicants with no perception of light, their place would be in Class I, the totally blind.

It is only by means of some such standard, based on angular measurements, that we can deal satisfactorily and fairly with applicants for industrial employment or for contemplated State aid, and I recommend the method described to those of my colleagues who are engaged in certifying blind applicants either for employment or for charity.

The Necessity for an Exact Definition of Blindness.

By N. BISHOP HARMAN, F.R.C.S.

THERE is only one possible exact definition of blindness, and that is to be found in any dictionary. In two standard works I find "blindness" is defined as "want of sight," and that is the one and only possible definition that can be given with any exactitude. If a man has sight of any degree, even though it be only perception of light, then he is not strictly blind; if there be no perception of light, then he is blind.

In our work we are, however, accustomed to extend the term to cases of partial blindness, and it is in these cases the difficulty arises. The demand indicated in the title of this discussion voices a desire on the part of many to arrive at some limitation of this looser application of the term "blindness" to varying degrees of partial blindness. The demand does not arise from any academic desire to furnish interesting definitions, but because of the public interest aroused by the proposed State aid or pension for the blind. Those who are interested in these matters, and who are lacking in professional knowledge, wish us to tell them who are to be counted blind and who are not.

So soon as we come to review these many cases of partial blindness, the extreme difficulty — nay, the impossibility — of arriving at any really useful definition, so far as it relates to the blindness itself, becomes apparent. Vision is a matter of several factors: acuity, field, mobility, mentality, the varying effects of light both night and day, and besides all these habit and custom. By no possible phrasing can we present a sufficiently simple definition that will embrace the possible variations in these several factors that make up sight, and state that such and such a variation shall be accounted blindness. The man who has a defect of the macula of each eye may be unable to read $\frac{1}{60}$ under any circumstances; yet, his visual field being otherwise perfect, he will be as free in his movements and as safe as the best sighted of his colleagues. But the man with a pinhole field and a high degree of visual acuity may be as helpless and as dependent on the guidance of others as the man who is totally blind. Again, there are cases where there is but small fault in the eyes themselves, but defect in the

development of the external muscles of the eyes ; the lack of mobility consequent therefrom presents as grave a handicap as any of these other defects. There are those whose mental condition affects their vision : at one time, under favouring conditions, they may obtain vision of all forms that would remove them from any strict definition of partial blindness ; yet at others they may fail to obtain any satisfactory vision. Lastly, there is the influence of habit. To those who are born with grave defect of vision, or who have sustained the loss within the first few months of life, any remnant of vision they retain is of the greatest value ; habit and skill in the use of their poor eyes, and the judgment of what their feeble impressions mean, are such that they are able to act with considerable freedom. On the other hand, to those who have sustained loss of sight of no greater degree, or even of less degree, in adult years the suddenness of the change leaves them as helpless as though they were completely blinded.

These variations in the conditions and effects of partial blindness render it impossible to gather them up into any sufficiently simple and intelligible formula. But suppose, for the sake of argument, it were possible for us to agree on any such formula. Would the definition present any advantages ? Such a definition would apparently have to state a minimum of acuity, of field, and so forth. When we have examined the eyes of a patient and have ascertained the standard of vision attained, can we say that we have gained any adequate judgment of the value of the sight the patient possesses ? I do not think that this can be affirmed. What we have got are a number of observations, but as they stand they are quite unrelated to life, that is, to the life of the patient, and accordingly they cannot be held to define his state of partial blindness. We have learned that he has a certain visual acuity, a field, and so forth, at a certain time and place, and under conditions of lighting, of mental liveliness, and general health, existing at that time, and probably never likely to recur at any other time. These findings are definite enough for the particular occasion, but they are no indication of the patient's disability in obtaining a livelihood.

Again, let us suppose that we could agree upon some definition. To what use would it be put ? This definition is asked for by laymen in pursuance of certain projected schemes of legislation. It follows, therefore, that our definition would have to come before Parliament as the considered opinion of a body of experts. It would thereon be subjected to the clever criticism of a number of acute minds bent on

favouring one or other extreme of the cause advocated. Every one of these skilled debaters would fall foul of our definition, and shatter its *ex cathedra* utterance by citing particular cases, "known to themselves," of patients who having less vision than that specified in our definition have attained brilliant success in ordinary walks of life; or, on the contrary, others would cite cases, also "well known to themselves," of folk who in their judgment were hopelessly incapable, and yet would be excluded from the embrace of this harsh and "unconscionable definition."

Supposing, for the sake of argument, that our definition did come before the august body of the Legislature. It is quite likely that it would emerge therefrom in so altered a form that we should wish we had never essayed our task, for the change might be in our opinion for the worse and not for the better. This is no mere speculation, it is based upon real personal experience. In 1910, on the invitation of the Gardener Trust for the Blind, I joined with Mr. Wilson, the Secretary of that Trust, in a deputation to the Registrar-General with reference to the collection of information regarding the blind by the census paper. We asked that in the census form of 1911 there might be a change from the older paper in which the entry was given under the unqualified term "blind"; and we asked that for the future the entry might read (1) "blind," (2) "partly blind." We added that if any definition of these terms were required (1) "blind" might read "totally or stone blind," with the explanation "not able to see his way about"; (2) "partly blind," (a) "a child who attends blind school," (b) "adult unable to earn living by ordinary sighted work." The view was expressed that most probably the terms themselves were sufficiently descriptive without added explanation. At the conclusion of our interview with the Registrar and his staff we were inclined to think that the object of the deputation had been attained. But, alas! when the actual census paper appeared, it was clear that we had failed and the first condition of the entry had been worsened, for it now appears as "totally blind." What effects this alteration was likely to produce on the value of the census of the blind for 1911 I have already indicated, for I was able to obtain the census returns of most of the blind children under my care in London schools, and to compare these returns with the actual state of their vision.¹

There is yet one other consideration that makes it highly undesirable

¹ *Brit. Med. Journ.*, 1911, ii, p. 520.

that we should ask Parliament to incorporate any hard and fast definition of blindness in any Act. When once a Bill has passed into law it is uncommonly difficult to obtain any modification of its terms. It is far better in such matters to secure such a general definition that the actual meaning of it shall be in the common-sense of the community and those who have to do with the working of the Act. Our ideas, and those of the community, are always in a state of flux, and with a general definition there is room for variation within reasonable bounds.

Again, would "an exact definition" be of service to those of our own profession who in subsequent years will have to examine the bad-sighted and state whether or no they come within the embrace of this definition? Any definition, to be a definition, must be exact, and the more perfect the exactitude the harder and faster the line of cleavage. In the first place, the task of accurately setting the cases on the one side or other of this line would present insuperable difficulties. In many cases our findings are so much more a matter of judgment than of rigid fact; and an unconscious bias in registering these findings would inevitably influence the manner of their record. The definition might be exact, but the manner of its usage would not be; it would be much more a matter of the good judgment or common-sense of the observer. This is a point to which I shall wish to refer again in making my recommendation for a definition of blindness.

From these speculations I will pass to the consideration of certain actual happenings that should be a good guide to us in the matter under discussion.

Blindness, in a certain section of the community, has already been defined by Act of Parliament, 56 and 57 Vict., Ch. 42, September 12, 1893. "An Act to make better Provision for the Elementary Education of Blind and Deaf Children in England and Wales" reads in Section 15 thus: "In this Act the expression 'blind' means too blind to be able to read the ordinary school books used by children." That definition for its judgment is, to my thinking, the definition of a Solomon. It relates the blindness to life. It recognises the use vision is to be put to in school, and states that blindness shall be considered to be the incapacity to perform the usual work of the school. I know quite well that the definition has been attacked, but the attack is purely academic, and bears no real relation to the facts of school life. It is said that there is no statement as to the nature of "ordinary school books," and that nothing is said as to the distance at which the reading must be

done. The very absence of these desired limitations proves to me the wisdom of the definition. We have only recently begun to understand what are suitable books for children's use, and the latest recommendations have been incorporated in the report of a Committee of the British Association. The recommendations differ from older views, and no doubt future views will differ again from these. But the terms of the Act cover all these variations. As to the point of the distance at which the book must be read, our legislators appeared to have some sense of the intelligence of the magistracy with whom the final and particular interpretation of the definition must rest. During the past twelve years I have had occasion to certify as blind, or within the meaning of this Act, no fewer than 1,500 children, and in those few cases in which I have had to support my certificate in the courts of justice, these alleged difficulties have never been apparent.

Again, it is objected by some that the definition allows of wide variations in the certification of cases of blindness. It is said that children in one district are certified as blind who would not be so certified in other districts. My experience does not lead me to attach any weight to this objection. Many of the children whom I have certified as blind have been transferred to blind schools, both in London and in the country, which are under the care of other ophthalmic surgeons, and in no case has there been a refusal to accept the child on account of any disagreement with the certification of blindness. Further, in my London schools I have had to receive many children who have first been entered at country schools, and in no case have I found it necessary to disagree with the original certification. That experience seems to me to be conclusive. The judgment of the surgeons who make the certifications is equal, even though they are not guided by the inelastic scale of an "exact definition."

In any effort to obtain legislative action precedent exerts a large influence. It is common to decry precedent; but in practice it has the advantage of being the path of least resistance, whilst if the precedent be a good one the advantage is manifest. There is the precedent of the definition of blindness in school children, and as has been indicated it is found good after twenty-one years of experience—it has attained its majority. But there are other precedents from which we may take guidance. Certain people have to be certified under the lunacy laws, and in the last year there has been a large extension of the application of the principle underlying these laws by the passing of the Mental Deficiency Act. Certification under these Acts is very

carefully safeguarded, for the very good reason that the laity are fearful that without such safeguards the liberty of the subject, and perhaps their own individual liberty, might be jeopardised. In these Acts there is no exact definition of the madness or lack of mind of these unfortunates; the whole reference is to the facts of life, as these affect them or others in relation to them. Idiots are those who are "unable to guard themselves against common physical dangers." Imbeciles those who "are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so." Feeble-minded those who "require care, supervision, and control for their own protection, or for the protection of others, or in the case of children those who appear to be permanently incapable of receiving instruction in ordinary schools." Some of those terms might be deemed dangerously vague, for there are a good many of us who "require care, supervision and control." But this danger is avoided not by an attempt at exact definition, but by bringing into play the lively judgment of personality. The certification has to be made independently by two medical men who are recognised by the proper authority as having knowledge of the matter on which they certify. One is reminded of the saying, "Out of the mouths of two witnesses every word shall be established"; the personal equation is a better safeguard than a rigid definition.

Following the lines of the practice established by Act of Parliament in the certification of blind children, and again in the case of the mentally deficient, I would suggest that any definition of blindness that we may recommend should have reference to the facts of life rather than to any data concerning degrees of visual acuity and the like. Our recommendation should take some such form as one of these: "Blind for the practical purposes of life," or "blindness of such seriousness as prevents earning a living wage," or, in the case of those who have lost their sight in adult years, "formerly sighted and become so blind as to be incapacitated from following former occupation." The actual phrasing of such definitions will be best determined after critical handling by ourselves and by laymen who have knowledge of labour conditions. We might even go so far as to recommend the adoption, with necessary alterations, of such a form as is now in use for the certification of the mentally defective. The new certificate would read as follows:—

£ County.

PENSIONS FOR BLIND ACT, 19 . .
MEDICAL CERTIFICATE.

In the matter of _____ of _____
in the County of X , an alleged blind person.

I, the undersigned _____ do hereby certify as follows:—

1. I am a person registered under the Medical Acts, and I am in the actual practice of the medical profession and approved by the Local Authority for the County of X for the purpose of giving medical certificates under the above Act.

2. On the _____ day of _____ at _____
_____ in the County of X , separately from any other practitioner, I personally examined the said _____ and satisfied myself that (s)he was a blind person within the meaning of the Act.

3. I formed this conclusion on the following grounds, viz.:—

(a) Facts observed by myself—

(i) at the time of examination.

(ii) previously to examination on _____

(b) Facts communicated by others—

Dated _____

(Signed) _____

Address _____

Under such a scheme as this each case would be examined by two medical men independently, the examiners would be practitioners approved by the authorities for this purpose, each would certify his opinion of the case and the facts upon which he based his opinion. Finally, the certificates might be subject to the critical examination of the medical officer of the acting authority. There would be check and counter-check. The certificates would be furnished by men resident in the district of the patient; they would know the circumstances of his work, the conditions of employment in the district, and their two certificates, with the data on which they based their opinion, would be

collated by the medical officer of the authority. It would be difficult to secure a better safeguard for the public purse. And it may be argued that the State would be satisfied with the security of double certification with the added check of its own officer, even as it is in the case of consigning one mentally incapable to permanent custody.

Finally, whatever the Section may determine with regard to this matter of the definition of blindness, so far as it affects the adult population, it is to be hoped that there may be nothing in any agreed definition that will weaken the security of the definition of blindness as affecting school children. That definition is established by custom, and it works well; it is scarcely possible to better it, and to qualify it or overshadow it by any other definition might prove a loss rather than a gain.

Mr. F. RICHARDSON CROSS said that after the admirable papers which had been contributed to the Section, he did not feel he could add much. But this question of the blind would become a legislative one, and Mr. Rockliffe had shown how working men themselves had been agitating for more to be done for these afflicted people. He thought it quite right that legislative notice should be taken of the matter; but there seemed in certain quarters a lack of proper recognition of what had already been done for the blind, and a somewhat antagonistic attitude towards the work both of individuals and blind institutions.

The education of the blind child had been placed on a sound footing. If, owing to defect of sight, a child was not fit for an ordinary school, some other arrangements must be made for him. There was the blind school and the intermediate school for myopes.

Mr. Grimsdale had been appointed member of a Committee before whom, probably, a projected Parliamentary Bill would be considered, in which the word "blind" constantly occurred, and the question must arise as to what "blind" really meant. Ophthalmic surgeons should have some clear and, if possible, similar ideas on the subject.

Much diversity of opinion existed as to what constituted blindness. For example, instructions employed by the Census Bureau for the securing of data for the twelfth Census of the United States (1900) required the enumerator "to ask whether all persons have *good sight* and good hearing—i.e., can see and hear well." If it was found that some member of the family *could not see well*, the enumerator was then

required to ask "whether he or she could see good enough to read a book; and should it appear that the sight is so seriously impaired that it is impossible for the person to read a book even with the aid of glasses, then you will note such person as BLIND, even though, as a matter of fact, he or she might have some power of sight."

In 1910 the Registrar-General in London, seeing that results of census inquiries and the figures regarding the number of the blind had not been satisfactory, was considering various proposals made for improving these figures by the introduction of a definition of "blindness" for use in census returns. The speaker was largely responsible for recommending that for some census purposes two headings of blindness would be useful: (A) "*Blind*, not able to count fingers in the daylight more than 3 ft. distance; (B) "*Partially* (economically) *blind*, cannot be educated, or gain a living by means of the sight." But that if only one class was registered in the census, "blind" should be defined as under (A). He thought the Registrar-General might be willing to make two classes if they could be sufficiently defined—but not more than two. We could not consider as blind only those who were "stone-blind," or had mere perception of light. All in Class (A) above were blind for all practical purposes, or, in other words, "deprived of sight." Class (B) had a greater or less obscurity in vision, so as to be unable to perform work for which sight was required. There would be a large number of cases of defective eyesight whose capacity for sighted labour could only be judged of by an ophthalmic expert. But he did not hold the view that laymen were in no way capable of judging who were blind and who were not. There was a certain group of cases about whom there could be no doubt.

At the present time in the workshops of the Blind Institution of Bristol there were eighty-seven workers; of these forty-eight were stone-blind, and thirteen had bare perception of light. Eight men and six women had $\frac{2}{60}$ of vision or more, and some of these see too well to be there. The other twelve count fingers from 1 ft. to 4 ft.

Snellen's test $\frac{1}{60}$ is about equivalent to counting fingers at 4 ft. A normal eye using a $-10D.$ or a $+10D.$ lens has a reduction of vision to about $\frac{1}{60}$, and is able to see dimly a door, or objects about the room, and, perhaps, could hardly be considered as blind. In low degrees of sight he regarded finger-counting as a more practical test than Snellen's letters. Counting fingers at 1 m. was a lower standard than $\frac{1}{60}$. For a start and for practical purposes one who could not count fingers beyond a yard might be classed as blind; those who could do so, but were unable to

earn a livelihood by reason of a deprivation or want of sight might be judged as economically blind.

In an attempt to put the matter on a sound basis for the earlier stages, we might, perhaps, be content with comparative inaccuracy, certainly with something short of scientific precision. Many of the difficulties would subsequently be cleared up by experience. Should the State give aid to the blind there would be a large increase of those applying for assistance, and it would be necessary to exclude those seeing too well. This must be left for ophthalmic experts. Many people who desired to live easily without work would make a claim for assistance; while the independent, spirited workman would endeavour to remain under employment, and would accept any position among his former mates rather than go to an institution, and be classed as a blind man.

As one interested in a large public institution for the blind, he looked forward to State aid and co-operation of the local authorities, in addition to charitable assistance, in order to improve, as far as possible, the condition of these afflicted people.

Mr. W. T. HOLMES SPICER considered that the matter had been thoroughly threshed out in the discussion: he had little to add; he agreed with Mr. Rockliffe and Mr. Cross in what they had said. The subject bristled with difficulties, and regulations could not be made which would be universally satisfactory. If acuity of vision were specified without mention of the size of the visual fields trouble would sometimes arise. But it would be well if, at some future time, a more exact definition of blindness were attempted, as a guide for those people who had to decide who were blind. That led to the next question — namely, whether lay members of a County Council were the proper judges of blindness; whether instead, the decision should not rest with ophthalmic surgeons, who were able to take a broad view of every case. He thought the Section should not be in a hurry to make definitions now, but should be content with signifying their willingness to help if applied to: and that any regulations they did make should be for the guidance of experts rather than for laymen.

The PRESIDENT (Mr. Priestley Smith, F.R.C.S.) reminded the Section that although they might be willing to advise the Government Departmental Committee, and might do so with advantage, their advice

had not been asked. In matters of public importance such as the measure proposed, the authorities, he thought, should refer to some recognised body of experts rather than to an individual. No one could deal with the question better than Mr. Grimsdale, who had their entire confidence, and if, at the beginning, the right course had been taken, he might now have been in a position to report to his Committee as an authorised representative of this Section.

Section of Ophthalmology.

February 3, 1915.

Mr. PRIESTLEY SMITH, F.R.C.S., President of the Section, in the Chair.

Case of Glass-worker's Cataract in a "Puddler" (Iron-smelter).

By BERNARD CRIDLAND, F.R.C.S.Ed.

M. F., AGED 50, has worked as a puddler for thirty years and has never been employed in the glass trade. There is no history of previous ocular trouble, and no family history of cataract. Vision has been failing for about a year, but especially during the past two months. Both lenses show the typical posterior polar and cortical opacities; they are rather denser in the left than the right. The eyes otherwise appear healthy.

DISCUSSION.

Mr. BERNARD CRIDLAND said that a puddler was exposed to glare for more hours per day than was the glass-worker and he could not, therefore, understand why the condition was not more common in iron-workers, and why it did not occur at an earlier age than in the case of the glass-worker. At the International Congress, when the "effects of light upon the eye" were discussed, he brought forward this point, but he could not then support it otherwise than by his recollection of cases. The present case came to him last month. Few iron-workers seemed to wear protective goggles. The question was whether puddlers should not be included in the Industrial Disease Schedule, as liable to suffer from this disease. If that were done, some other name seemed to be called for; "glass-worker's cataract" did not seem a scientific or sufficiently descriptive name. In America, where the condition was associated with working in iron, it was named "heat cataract." But it had not been decided whether the disease was produced by the heat rays or whether it was the light rays, or both combined. He therefore suggested the term "ray cataract." He had just been informed by Mr. Leslie Paton that he showed a case of X-ray cataract some time ago, and that would lend support to this suggested name, as the condition was the typical one from which glass-blowers suffered.

Mr. C. B. GOULDEN said he thought this type of cataract was seen in other cases in which men were exposed to a bright glare. He had at present under care a patient whose occupation was that of welder. He had in both eyes a posterior polar cataract very similar to that seen in glass-blowers. If an investigation were made as to the incidence of cataract among iron-workers, he thought the condition would be found to be much commoner than was now believed. But among welders the affection did not seem to be common. He knew of one, in a boiler works, and the man who preceded him also lost his sight, but he could not be traced by the speaker. A remarkable feature about the subjects of these posterior polar cataracts was that a long time elapsed before they complained that defective sight prevented them from working. It would be found that their pupils, in ordinary daylight, were much larger than in ordinary people, so they had a chance of seeing round their cataract. In the case of a glass-worker he had seen, in spite of a posterior polar cataract which had been diagnosed years before, he still had full vision with proper correction.

Dr. LEGGE (Home Office) said he understood that Mr. Cridland had visited a number of iron-works in the Midlands to investigate this condition, and had examined the lenses of a number of workers. He would like to know, approximately, how many men he had so examined, and in what proportion he found the condition present. He asked because it was necessary to proceed tentatively in scheduling diseases under the Workmen's Compensation Act. In the case of glass-workers' cataract, in connexion with which the evidence was very strong, he examined the lenses of 513 glass-workers, and found sixty-four with the lens affected in the right eye and seventy-two in the left, generally of the posterior cortical type, exactly as in the patient shown. The evidence was so strong as to leave no doubt on the question of scheduling the condition. But if on examining 500 iron-moulders only two or three were found to be suffering from the condition, it would not be so easy to schedule it. A similar remark applied to the interesting case of a welder which Mr. Goulden described. He asked whether Mr. Goulden could describe what form of welding was done by that man; whether the oxyhydrogen, the acetylene, or the electric form of welding.

Mr. GOULDEN: The ordinary furnace welding.

Mr. J. HERBERT PARSONS said this case interested him because, two or three years ago, as Secretary of the Royal Society Committee on glass-workers' cataract, he sent out a series of letters to the ophthalmic surgeons in the iron-working districts, in order to find out whether the condition was prevalent amongst iron-workers. The replies he received were uniformly negative. It showed the value, or the absence of value, of such inquiries. The present patient showed the typical condition found in glass-workers' cataract. He doubted, though probably Dr. Legge would have better information on that point, whether these men looked into the furnace more than did

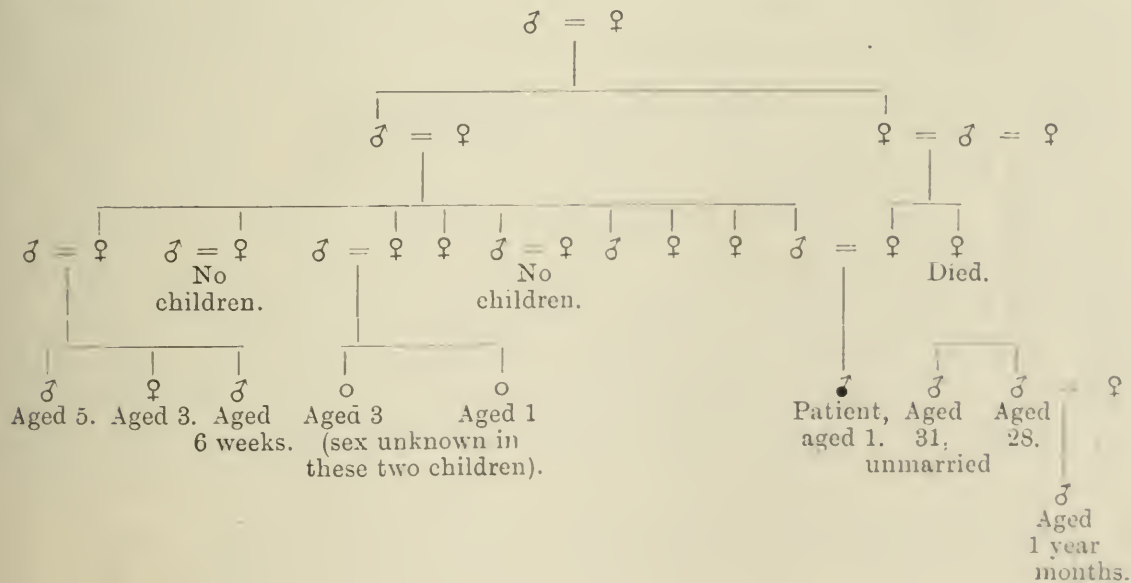
glass-workers. The type of glass-worker who mostly suffered from it was the finisher, who usually was a man who had served a long time at the craft; he was perpetually looking into the furnace or at the ring of molten glass which he put on the top of the bottle. He was under the impression that iron-workers protected their eyes by means of tinted glass as a rule, when looking into the furnace. He did not know whether that practice was universal. The Royal Society Committee was not yet defunct, and certainly it had not finished its work. At present they were engaged in investigating the heat problem. He was not sure whether it was wise at the present time to alter the name of the condition. He preferred to adhere to the name "glass-workers' cataract" even though the condition occurred also in iron-workers; at all events until there was more certainty as to the exact pathogenesis. The Royal Society Committee would be very much obliged by any information which Mr. Cridland or other members practising in iron-working districts could give.

The PRESIDENT (Mr. Priestley Smith, F.R.C.S.) suggested that an inclusive term like "furnace-workers' cataract" might be applied. Furnace work was common to both industries, and that term did not suggest what particular rays were the causal ones.

Amaurotic Family Idiocy in an English Child.

By E. A. COCKAYNE, M.D., and JOHN ATTLEE, M.D.

B. B., MALE child, aged 1, the only child of healthy parents, who are first cousins. They are of English descent. No similar disease has occurred in any other member of the family.



The child was quite healthy, sitting up and taking notice of his surroundings, until aged 5 months. He then began to take less interest and became gradually weaker. He is still well nourished, but shows marked weakness of all the muscles, especially those of the neck and back. Pes cavus is present. The knee-jerks are present and equal; the abdominal reflexes are absent. Eyes: There is slight lateral nystagmus. Pupils equal and react to light. Fundi show the macular changes characteristic of Tay-Sachs disease. There is no optic atrophy.

DISCUSSION.

Dr. COCKAYNE said that the case was brought in view of Dr. Batten's and Mr. Mayou's paper. The child was of English descent, whereas nearly all the recorded cases of the condition had been in Jews of foreign extraction. When affecting English children, the patients were usually older. The present patient had the Jewish type of the disease. There was no other case in this family. The parents were first cousins.

Dr. ATTLEE said the case was absolutely typical of the kind described in 1889 by Mr. Waren Tay in the *Ophthalmological Society's Transactions*; indeed, Mr. Tay's picture might have been taken from this case.

Spontaneous Rupture of the Cornea in Secondary Glaucoma caused by Dislocation of the Lens.

By N. BISHOP HARMAN, F.R.C.S.

THE patient whose eye is here exhibited was seen by me in March, 1911. He is a blacksmith by trade, and stated that about eight months previously he fell down, sustaining a bruise over the right temporal region; the eye was red and painful for some time after. At the first examination the eye was very hard, tension plus 3, the pupil widely dilated, and the iris reduced to a rim 1 mm. broad; eserine had no action upon it. The cornea was quite clear, so that the interior of the eye was clearly seen. The dislocated lens could be seen floating within the posterior chamber towards the nasal side. The disk was deeply excavated, quite pearly in colour, and there was no perception of light. The left eye also was defective. The pupil was irregular in contour but active to light, the iris was tremulous. A mydriatic acted well on the pupil, and the lens was then seen to be slightly dislocated. The fundus was normal, and vision $\frac{6}{24}$.

Probably this man always had some dislocation of his lenses, but in the right eye the condition had been aggravated by injury. No doubt the glaucoma had been set up by the irritation of the swinging lens.

The man was seen on several occasions in the years 1911, 1912 and 1913, indeed, he came up frequently for class demonstrations. There was no change in his state until February, 1914. In that month he came up with the eye bandaged and stated that blood suddenly gushed out of the eye when he was walking the streets. The lower part of the cornea was found to be ruptured horizontally, and the membranes were protruding from the gap. The eye was at once excised. Section of the eye shows that there was an expulsive retro-choroidal hæmorrhage which had driven the membranes forwards, and these are stretched between the disk and the cornea, and hang out from the rupture. The eye was full of blood except for the stretched membranes.

The man was quite sure he had not knocked the eye, and so far as he could remember there had been no sudden strain, neither had he felt any pain before the sudden rush of blood. The last visit to me prior to the rupture of the cornea dated three months back. At that time there was no sign of any ulceration of the surface, but the cornea was completely anæsthetic. Neither were there any cracks in Descemet's membrane such as are so common in buphthalmia. Three months is, however, plenty of time to allow of degenerative changes to have taken place in the membrane, and so permit of the apparently spontaneous rupture. Without some such change as this it would be difficult to account for the incident.

Granuloma of the Iris.¹

By N. BISHOP HARMAN, F.R.C.S.

THE patient was shown to the Section in 1913. She was then aged 32. There was a small rich brown mass at the base of the lower part of the iris of the right eye. The rest of the iris was brown with green at the deeper parts. A few spots of k.p. were to be seen on the cornea, and a small leash of vessels at the limbus over against the

¹ Sequel to case exhibited on June 4, 1913, under the title of "Melanotic Growth of the Iris," *Proc. Roy. Soc. Med.*, 1913, vi (Ophth. Sect.), p. 106.

growth in the iris. When the pupil was dilated the border was not retracted in the region of the growth. Nothing abnormal could be seen within the eye; the vision with a small correcting glass was $\frac{6}{6}$.

The case excited considerable attention. My own view was that it was a sarcoma, and that was the general opinion of the Section; an enucleation was advised. A suggestion made in the general talk about the case that the eye should be left two or three months and events noted was considered unjustifiable. After the meeting was over I found there was no need to recommend the patient to sacrifice the eye, for she and her mother had heard so much that they were only too anxious to get rid of the eye.

The patient was admitted to hospital under a colleague, and he, on getting the report of the case, agreed with the diagnosis and enucleated the eye. The eye was at once sent to Mr. Affleck Greeves in Zenker's fluid. The following is his pathological report.

Pathological Report by Mr. Affleck Greeves.—Granuloma of iris: The eye was fixed in Zenker's fluid, frozen, and divided. A swelling projects $\frac{1}{2}$ mm. from the anterior surface of the iris near its root. No projection from posterior surface. Microscopically, the swelling is a granuloma, on the anterior surface of which the iris stroma pigment appears to have proliferated; the main mass is made up of groups of epithelial cells, surrounded by lymphocytes and plasma cells, intersected by lines of enormously enlarged chromatophores. There are no giant cells. Some isolated nodules of plasma cell and lymphocyte infiltration are present in the iris. The ciliary body, on the side of the granuloma, shows similar nodules. No necrotic areas are present in the granuloma.

The case points to the advisability of taking the Wassermann reaction even when there is not the slightest suspicion that the diagnosis of malignant disease is not correct. There was no suspicion in this case, yet the pathological finding proved that the diagnosis was wrong. Before the eye was removed I had seen the mother, the husband, and two of the four children of the patient. There was no hint of any syphilitic taint. Since the operation the Wassermann reaction has been taken. The patient gave a "partial positive," and three of the children a similar reaction; one only gave a negative reaction.

Sarcoma of the Choroid.¹

By N. BISHOP HARMAN, F.R.C.S.

THE full details of this case, with a drawing of the fundus, were given in the notes published under date of exhibition. The woman was of mature age and had recently found she could not see out of one eye. Just above and partly obscuring the disk a hemispherical mass was found, bordered above by many hæmorrhages. The mass was thought to be a sarcoma, and enucleation of the eye proposed. After the experience of the case just reported the Wassermann reaction was taken and found negative. The pathological report by Mr. Affleck Greeves proves that the diagnosis was correct.

Pathological Report by Mr. Affleck Greeves.—Sarcoma of choroid : The eye was fixed in Zenker's fluid, frozen, and divided. In the upper and posterior quadrant a colourless growth, about as large as a pea, was found. The retina was detached over the growth, but not elsewhere. Microscopically, the growth springs from the choroid. It is apparently quite unpigmented and consists of closely packed spindle-shaped cells. The retina is not adherent to the growth. In front there is a small simple detachment of the retina, and in the space between retina and choroid is a hæmorrhage (seen clinically). The growth is a typical spindle-celled sarcoma, only remarkable because of the absence of pigment, and because it is less exuberantly vascular than the usual sarcoma of the choroid. The anterior parts of the eye are normal. No invasion of sclera or optic nerve.

A Trench Periscope.

Shown by PRIESTLEY SMITH, F.R.C.S.

THIS consists of a wooden rod 1 in. square and 4 ft. long and two mirrors. When in use the rod carries the mirrors, one at the top and the other as far below it as may be necessary for safety. The nearer the two mirrors can with safety be brought together the larger the field

¹ Sequel to case exhibited on November 5, 1913, under the title of "Tumour of the Choroid," *Proc. Roy. Soc. Med.*, 1914, vii (Ophth. Sect.), p. 8.

obtained. The mirrors are 5 in. wide and $3\frac{1}{2}$ in. from top to bottom: they are mounted in sheet brass frames with square sockets. By means of the sockets they are mounted on the rod when in use. The position of the lower mirror is fixed by holes and a peg. When not in use the mirrors are placed in a box, which also contains some spare mirrors. The rod stands between the lower mirror and the observer's face, but does not interfere with his view of the mirror when the instrument is held near to the face. Field glasses can be used with it, one barrel standing on each side of the rod.

Lieut.-Colonel ELLIOT, I.M.S., said that all periscopes could be divided into two classes, the binocular and the monocular. From conversations with officers who had returned from the front, it appeared that the periscope was of little use unless it was binocular, because the enemy were now sapping to within 10 or 15 yards of the trenches held by our men, and the officer needed to know how near they were when they made a movement. He did not see how that could be known unless the periscope was binocular. He thought that some of our trenches had been rushed because the signal had been given too late; the enemy were on our men before they were ready.

Family Cerebral Degeneration with Macular Changes.

By F. E. BATTEN, M.D., and M. S. MAYOU, F.R.C.S.

INTRODUCTION.

THE purpose of this communication is to put before the Section the pathological results which we have found in the eyes and central nervous system of two members of a family affected by the condition which has been described under the following titles: "Family Cerebral Degeneration with Macular Change" (Batten and Mayou); "Maculo-cerebral Degeneration (Familial)" (Oatman); "Progressive Familial Macular Degeneration" (Darier); "Late Infantile and Juvenile Form of Amaurotic Idiocy" (Vogt, Bielschowsky, and others). It is not our purpose to go into the literature of the subject, nor to attempt to separate into groups the variety of cases which have been described under the above and like headings, this having in part been done in a recent paper by H. Gifford, but the following short review is necessary for understanding the present position of this disease.

In 1897 R. D. Batten described symmetrical changes in the fundus consisting of an aggregation of fine stippled pigment at the macula,

with slight pallor of the optic disk in two brothers, aged 14 and 21 respectively. The sight began to fail at the age of 14. There was no mental defect.

In 1903 F. E. Batten described symmetrical changes in the maculae in two members of a family with cerebral degeneration starting about the age of 6.

In 1904 M. S. Mayou described cerebral degeneration with symmetrical changes in the macula in three members of a family starting about the age of 7.

In 1904 Hirschberg recorded a similar condition with mental symptoms in a boy aged 14.

In 1907 Oatman showed three cases affected with maculo-cerebral degeneration, and in 1911 published a detailed description of the condition.

In 1908 Nettleship read a paper on cases possibly allied to Tay's infantile retinitis.

In 1910 Rogalski reported the case of a girl, normal till the seventh year, when mental powers began to fail, eyesight began to fail at the age of 10, and the macula showed a pigmentary degeneration. The patient died when aged 26, and the microscopical examination of the nervous system is said to have shown changes similar to those found in the Waren Tay-Sachs cases. No other members of the family were affected.

In 1910 Behr reported the case of a girl, the fifth member of a family of eleven, whose sight began to fail when aged 14. Progressive dementia and paralysis followed, and death occurred when aged 23. An elder brother and younger sister were similarly affected at the same age.

In 1910 Kuffler reported four cases in a family of seven children. The disease manifested itself when the children were aged 6.

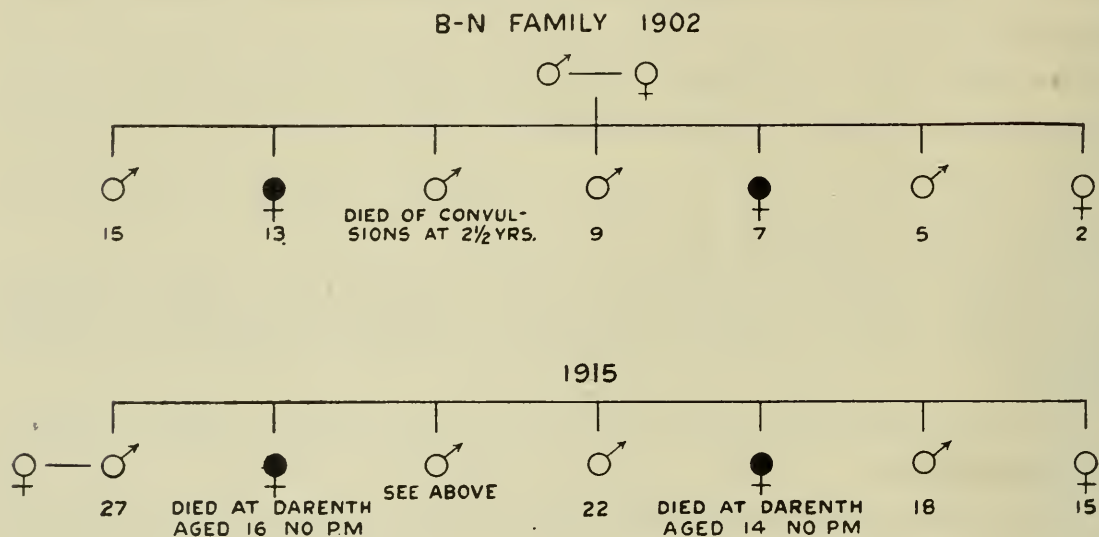
In 1912 Gifford reported one typical case in a family of seven, one other child being probably affected; he carefully analysed all the recorded cases and discusses the pathology on the basis of Schaffer's, Vogt's, Behr's, Rogalski's and Brook's pathological examination.

In 1914 Darier discussed the relationship of the various forms of progressive familial macular degeneration, and quotes some personal observations.

In 1914 F. E. Batten described the changes found in the central nervous system of a child affected with the disease.

The last three papers contain references to the literature on the subject.

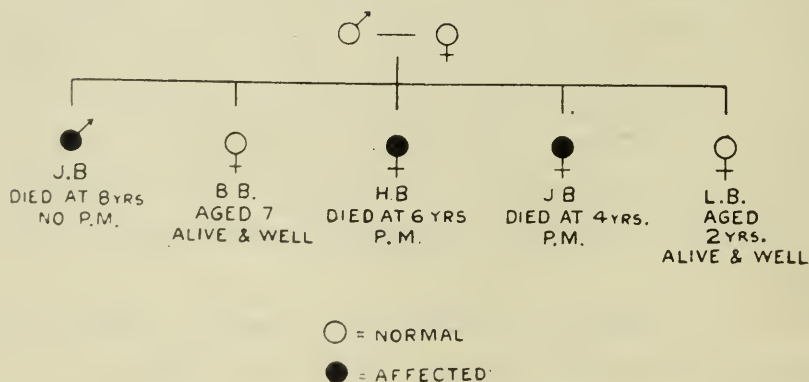
It may be well to take this opportunity to complete the history of the B—n family, the cases originally shown and described in 1903 before the Ophthalmological Society.



The two children died in Darenth Asylum in 1905 and 1909, but in neither case was an autopsy performed.

A letter from the father in 1915 states that his younger children are quite well and have never shown any symptoms such as those exhibited by his two elder girls. The elder son is married but has no children.

The family to be considered in this paper consists of five members—one male and four females:—



The mother and father are perfectly healthy and normal individuals, they are not related, and there is no history of mental defect in any of the collaterals. The mother has four brothers and four sisters; five of them are married and have children who are perfectly healthy; the father has one sister, married, but without children. The father had two brothers. One died of pneumonia, the other by accident, at the ages of 18 and 22 respectively.

The eldest child, a boy, James B., aged 3 years 10 months, was in May, 1909, admitted to the Hospital for Sick Children under the care of Mr. Waugh, suffering from epilepsy. It was stated that three months previously he was run over and suffered from a scalp wound on the vertex. He was unconscious, and was kept in the district hospital for three weeks after the accident. Since that time he had on various occasions suddenly fallen down and had convulsive movements of all his limbs. He has become irritable and constantly dirty in his habits since the accident; previously he was occasionally dirty. On August 10, 1909, Mr. Waugh trephined the boy and turned down an osteoplastic flap and exposed the whole area of the scar. The skull and dura mater appeared perfectly normal, and after opening the dura there appeared to be a slight excess of the subdural fluid, and the cortical veins were congested; otherwise nothing abnormal was noticeable. The boy recovered from the operation, but still continued to scream loudly and was in much the same condition as before the operation. No examination of the cerebrospinal fluid was made. He was discharged from the Hospital and died in February, 1913, when aged 8, having slowly passed into a demented condition. No autopsy was made.

The second member of the family, Bertha B., aged 7 in 1914, was physically and mentally a normal child, and on examination nothing abnormal could be discovered. She was said, however, to have had nocturnal incontinence.

The third member of the family, Hilda B., aged $5\frac{1}{2}$, was well until the age of $3\frac{1}{2}$. She was born at full term, in normal labour without instruments, cut her first tooth at the age of 5 months, walked at the age of 20 months, and began to talk about the same time. She had her first fit when aged $3\frac{1}{2}$, and since that age has had frequent fits, has gradually lost power in the limbs, has become mentally defective, dirty in her habits, and takes but little notice of her surroundings. On examination in June, 1913, she lay in bed with her legs extended; when placed in the erect position she stood on tiptoe, but could not walk. She failed to grasp objects with her hands, but could move her

arms in all directions; she was unable to talk, and had attacks in which she screamed loudly. The pupils reacted well to light, the child could follow a light, the optic disks were pale, the vessels were normal, and no change could be seen in the fundus. The knee-jerks were active, the plantars both gave extensor responses, abdominal reflexes were present. The cerebrospinal fluid was quite clear and normal in character. The Wassermann reaction was negative both in the blood and in the cerebrospinal fluid. The child remained in hospital for about four weeks, but practically showed no change during that time. The fits were controlled by small doses of hyoscine. The child was discharged from the hospital in June, 1913, and readmitted to hospital in February, 1914. She was then emaciated, had rigidity of arms and legs, took no notice, did not see, but the pupils still reacted to light. The optic atrophy was more marked, and at the maculæ there was a fine pigmentary degeneration with considerable disturbance of the pigment in the periphery of the fundus (for full description see pp. 75-78). The knee-jerks were brisk, ankle clonus was present, and both plantars gave an extensor response. The child died on March 7, 1914, and a post-mortem was made which showed a normally convoluted brain with shrunken convolutions. The microscopical appearance of the brain and eyes in this case will be dealt with later.

The fourth member of the family, Jessie B., aged 3 years 10 months, was said to be quite well up to six months previously. She was apparently a normal child in every respect till the age of 3; she learnt to walk and talk and was clean in her habits. Fits occurred about the age of 3 and recently had recurred with increasing frequency, and she had passed into a condition showing marked mental defect. She, like her sister, had frequent attacks of screaming, could just support her weight on her legs, but could not stand or walk. The pupils reacted quite well to light, the disk and fundus of the eye were quite normal. The limbs could be moved in all directions, the knee-jerks were active, there was no ankle clonus, both plantars showed an extensor response, and abdominal reflexes were active. The cerebrospinal fluid was perfectly normal and the Wassermann reaction was negative in both blood and cerebrospinal fluid. The child contracted chicken-pox and died in September, 1913, aged 4. A post-mortem examination was made, which showed a normally convoluted brain with but little shrinking.

The fifth member of the family, Lily B., aged $2\frac{1}{2}$, is alive and perfectly healthy. She talks and walks quite well for a child of her age.

The knee-jerks are present, and the optic disks and fundus are normal. The grandmother believes that this child is going to be affected because of a slight turning out of the left foot.

CLINICAL AND PATHOLOGICAL DESCRIPTION OF THE EYES OF
HILDA B. (BY M. S. M.).

Clinical Description.

The anterior part of the eye was normal; the pupils were dilated with homatropine and cocaine. The child had evidently lost all perception of light. On examination of the fundus the disk was a greyish-white colour with vessels of nearly normal size. On the macular region there was a diffuse pigmentation. The pigmentation extended to a more or less marked extent into the retina around, and in this situation and even right out to the periphery there was a very marked stippling of the pigment, more so than is usually seen in children.

Pathological Description.

The eyes were injected immediately after death with Zenker's fluid, and were removed as soon as possible and put into the same solution, the anterior halves being removed. The specimens were embedded in celloidin, cut, and stained by logwood eosin, and after removal of the celloidin by thionin blue. The sclera and choroid were practically normal and changes were confined to the retina. They varied considerably in their distribution. Near the disk, especially on the nasal side, the retina was normal in its appearance, except that the ganglion cells were diminished in number and showed signs of degeneration. The principal change was around the macular region and also in some situations in the periphery. In situations where degeneration was most varied there was a complete disappearance of the inner nuclear layer, ganglion cells and the rods and cones. In the nerve-fibre layer and in the position of the ganglion cells the retina was somewhat thickened and there was an increased number of darkly staining nuclei. In the retinal pigment layer the pigment had migrated from the cells to a slight extent into the outermost layers of the retina. The change, therefore, was one of disappearance of the neural elements of the retina with some slight increase in the proliferation of the supporting structures.

All stages of this degeneration up to a nearly normal retina could be traced in the specimen. The earliest sign of degeneration seemed to be a change in the ganglion cell layer, the cells of which become swollen and the Nissl granules disappear; the cytoplasm stains uniformly all over, except around the nucleus where there seems to be some condensation of the cytoplasm, vacuolation occurred, but was rare. In a



FIG. 1.

Cerebral degeneration associated with macular changes. The retina showing the extreme stages of the retinal degeneration. 1, disappearance of the ganglion cells, with thickening and proliferation of the nuclei of the inner layers of the retina; 2, disappearance of the inner nuclear layer and of the rods and cones; 3, migration of the retinal pigment.

very large number of the cells the nucleus itself had disappeared and the cells were merely represented by a cell containing uniform lightly staining cytoplasm (fig. 3).

The next change seemed to be a slight œdema in the internuclear layers, after which there was a gradual disappearance of the inner nuclear layer and associated with it a disappearance of the rods and

cones—if anything the latter going first (fig. 2). The pigmentation seems to be the final stage and only occurs after the disappearance of the rods and cones. To sum up the retinal change one would say that there is a disappearance of the neural elements starting first with the ganglion

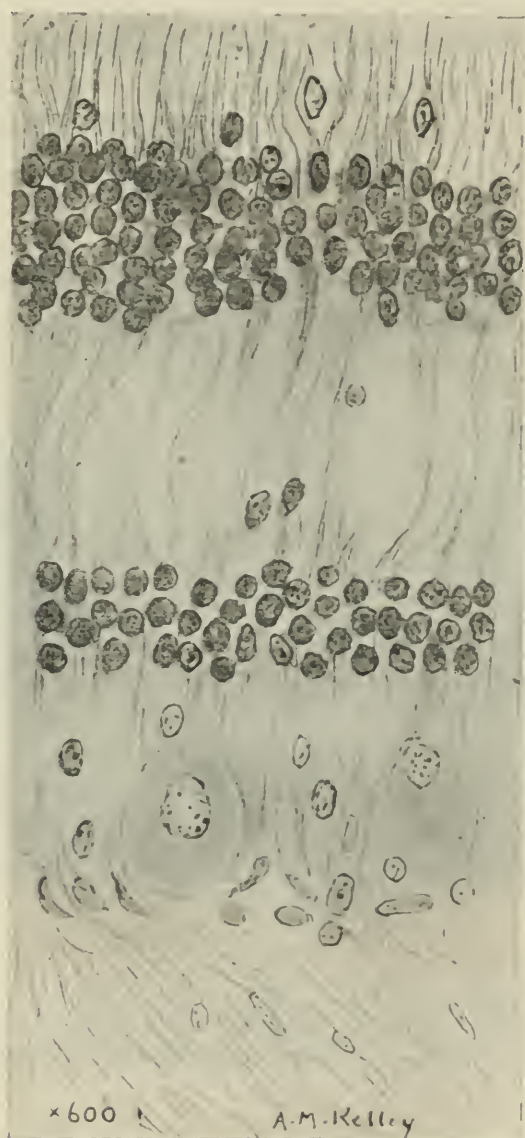


FIG. 2.

Cerebral degeneration associated with macular changes. Retina showing early signs of degeneration—oedema of the intranuclear layer and changes in the ganglion cells.

cells and ending finally with the disappearance of the inner nuclear layers, and rods and cones, with a slight increase of the proliferation of the supporting structures with their nuclei; and in the situation where

the change is extreme a tendency for the migration of the pigment from the pigment cell layer (fig. 1).

There is a close relationship between these cases and the cases of amaurotic family idiocy. The pathological changes which are found in ganglion cells of the retina in these cases of cerebral degeneration are similar to the changes which are found in the cases of amaurotic family idiocy. At first sight it is a little difficult to reconcile the two ophthalmoscopic pictures being associated with the same pathological changes. The only difference in the two diseases appears to be that the macular



FIG. 3.

1, normal ganglion cell of the retina; 2, 3, 4, degenerating ganglion cells in the retina; from a case of cerebral degeneration associated with macular changes.

region in amaurotic family idiocy shows a much more marked œdema in the internuclear layers and therefore a white area around the macula which is associated with a hole or thinning of the retina at the fovea due to the œdema. It is instructive to compare a typical case of family amaurotic idiocy, under the care of the late Mr. Hancock at the Children's Hospital, Shadwell, in which the fundus showed the usual red spot at the macula surrounded by a white area, with the present case. The brain and nervous system were examined by Dr. Mött and

an account was published in the *Archives of Neurology* (Claybury Laboratory), 1907, vol. iii, p. 232. The eyes were injected with formol immediately on the death of the child, and removed within an hour of death and placed in formalin. One cannot emphasise too strongly the importance of immediate fixation, as I have found that normal retinal ganglion cells begin to show post-mortem changes (vacuolation) six hours after death. The retina from one eye was removed and examined on a microscope slide on the flat. At the macula there appeared to be a hole, but closer examination showed that the limiting membranes of the retina were still present, although there were

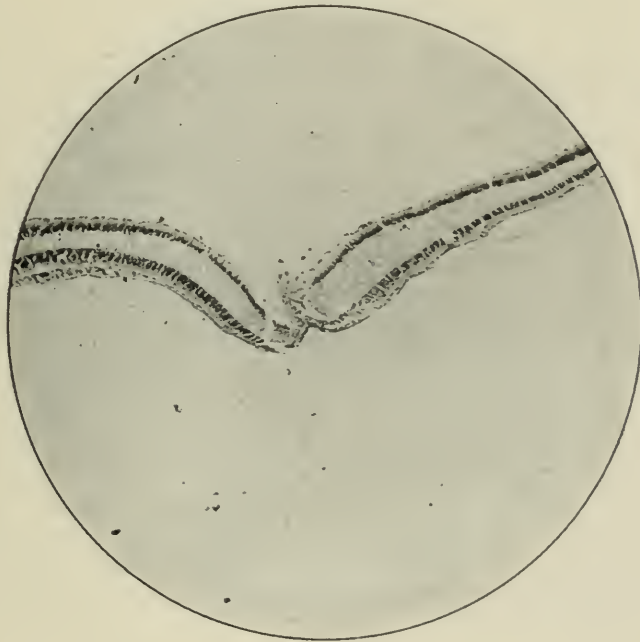


FIG. 4.

The retina at the macula from a case of amaurotic family idiocy, showing the œdema of the intranuclear layer around it.

practically no cellular elements. Sections were made of both retinae. The ganglion cells showed most marked change; they were swollen and the granules had disappeared, the cytoplasm had collected around the nuclei of the cell, leaving the remainder of the cell staining uniformly. In some the nuclei had disappeared entirely. Vacuolation of the cells was not common although it occasionally occurred; this has been a marked feature in some of the recorded cases. Around the macula there was a very marked œdema of the internuclear layers which probably correspond to the white area which

is seen during life. Away from the macula the retina was of fairly normal appearance, except that the ganglion cells were showing signs of degeneration. It will be seen, therefore, that the changes in the ganglion cells are similar to those which have already been described in the case of amaurotic family idiocy. The œdema of the internuclear layers is present in both diseases, but is very much more marked in the case of family amaurotic idiocy, and also in the case of amaurotic family idiocy the final degenerative changes do not make their appearance—that is to say, there is no pigmentation and the

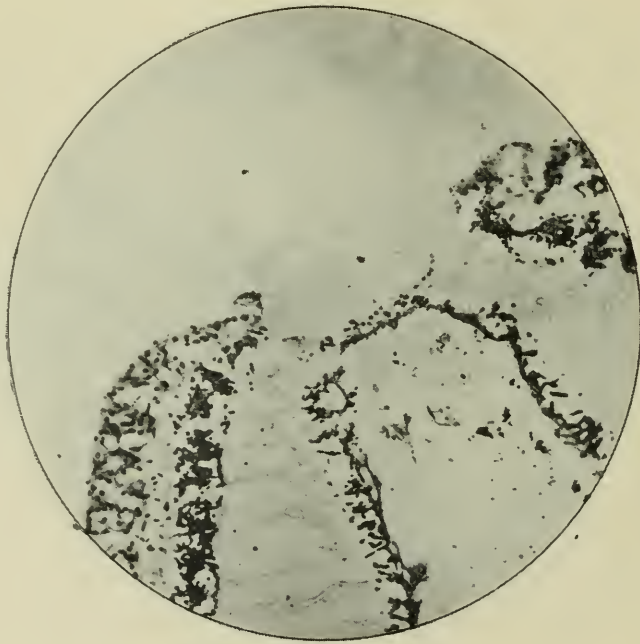


FIG. 5.

The macula from a case of amaurotic family idiocy, showing the limiting membranes present at the macula.

nuclear layers and rods and cones have not entirely disappeared. This, together with the fact that the œdema is more manifest, is probably due to the cases being of much shorter duration and the change a much more acute one, and the patient dying before the final stages of degeneration took place.

This view is also somewhat borne out by a case of cerebral degeneration associated with macular changes in an idiot boy, aged 15, recorded by Hirschberg in which the pigmentation was associated with a white area at the macula.

Pathological Examination.

Hilda B. died at the age of 6 after the disease had existed two and a half years. Brain: Weight, total 765 gm.—cerebrum 692 gm., cerebellum 73 gm. Appearance: There was some shrinking of the convolutions, but the arrangement of these was normal; otherwise there was no change in the nervous system to the naked eye.

The nervous system has been examined by Dr. H. J. Prentice, and the following is his report:—

Microscopical Examination.—Speaking generally, the outstanding feature throughout the central nervous system is the marked degree of



FIG. 6.

Degenerating retinal ganglion cells, from a case of amaurotic family idiocy.

disintegration of the cells, unaccompanied by any marked degeneration of the tracts.

Cerebrum.—Cortex: The cells appear diminished in number. There is widespread degeneration of the cells, which naturally is seen most strikingly in the Betz and large pyramidal cells. The cell bodies have lost their pyramidal shape, assuming a more or less globular form (fig. 7). The nucleus is displaced, usually towards the apex of the cell. The Nissl substance is disorganised; in some cells it appears collected towards the apex of the cell while the basal part of the cell is clear; in other cells both nucleus and Nissl substance have degenerated, and

only a fine diffuse granulation remains. In a comparatively early stage of degeneration the protoplasmic network becomes simplified in design, and very coarse. The fibrils are seen entering the cell from the dendrites, they then disappear in the swollen body of the cell, but reappear at the point of emergence of the axon. Many of the cells are so disintegrated that they appear as scarcely more than faintly outlined

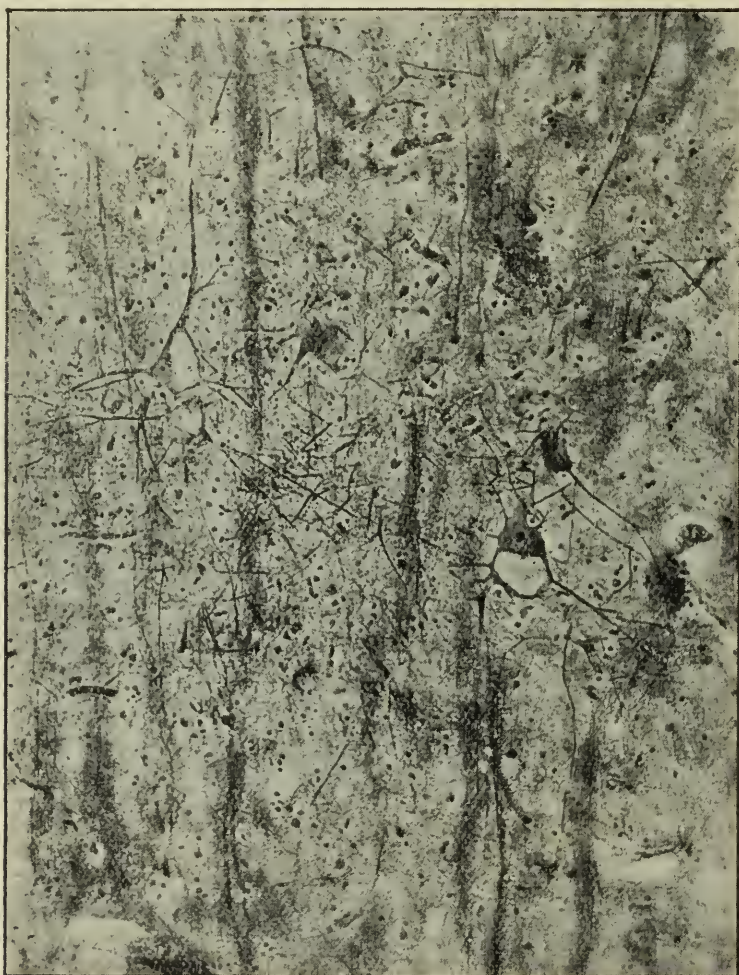


FIG. 7.

Section of cerebral cortex stained by Bielschowsky's method, showing swollen Betz cell with vacuolation.

spaces. When stained with Scharlach R the substance of the cells shows a finely granular red appearance. This is seen almost universally in the larger cells. In addition, in the perivascular lymphatics there are seen brilliantly red-stained masses of fat. In some regions, notably in the occipital lobe, there is very definite neuroglial overgrowth, mainly in the deeper layers of the cortex.

Cerebellum.—(1) Cells of Purkinje. The number is much diminished. Their position is in most cases normal, but there is a tendency for them to wander into a more superficial position; in many instances cells of Purkinje are seen almost on the surface. Very few can be considered normal in structure. Generally the cell bodies are swollen, the nucleus is displaced, and the protoplasm is diffusely granular. In many instances the nucleus has disappeared, and the protoplasm stains very poorly. The dendrites show two forms of degeneration: (a) They are generally swollen; (b) they have local, almost globular, swellings upon them,

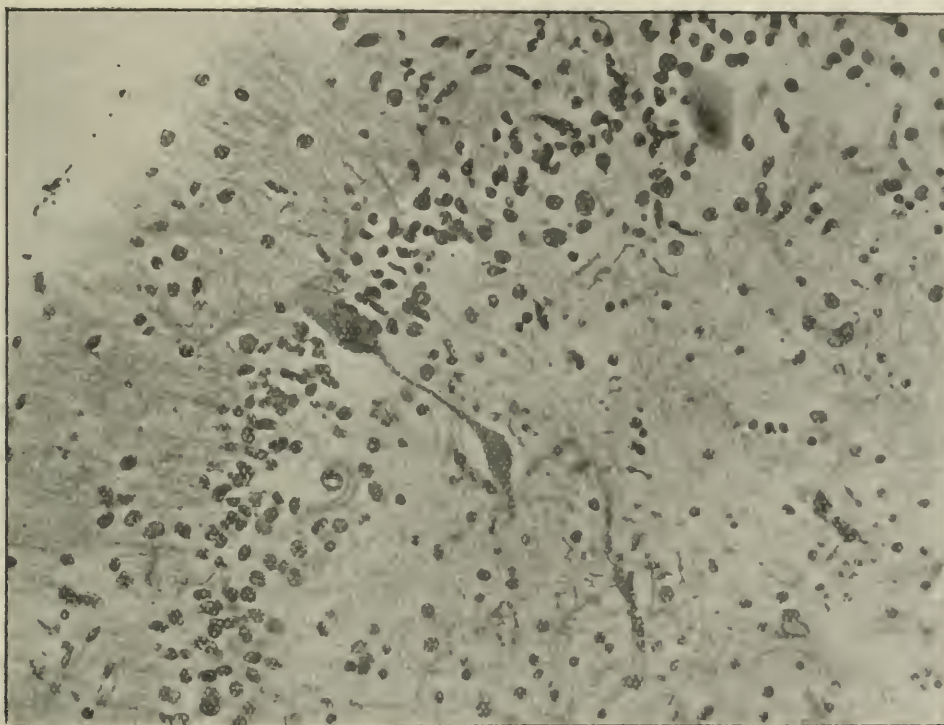


FIG. 8.

Section of cerebellum stained by the same method, showing degenerate Purkinje cells, with swelling in dendrite.

situated usually at a point of bifurcation. In both instances the fibrils can be seen as though separated from one another by the swollen ground substance (fig. 8). The axons do not show any general swelling, but in many instances fusiform or globular local swellings are seen upon them, situated usually about one and a half to two cell lengths from the base of the axon. As in the case of similar swellings on the dendrites, the fibrils are seen clearly separated by or spread over the swelling of the ground substance, they themselves not being thickened. Some axons appear to turn back and enter the molecular layer.

(2) Cells of the granular layer: The layer is much diminished in thickness, being only about one-third of the normal depth in most places. On examination under a high power it is seen that the number of typical granule cells in this layer is even smaller than appears to be the case under a low magnification. A large number of cells are present which have a diameter half as large again as the typical granule cell, a nucleus smaller relatively to the size of the cell, and a coarsely granular cytoplasm; in outline the cells are irregular. These cells make the granular layer appear thicker than it really is. Were they not present the layer would consist of not much more than a single layer of cells in many places.

(3) Dentate nucleus: The cells are small in number, they stain very poorly (both nucleus and cytoplasm), and many appear but as cell shadows.

(4) Fibres: There is marked absence of the basket arrangement of fibres around the cells of Purkinje. The whole white matter is small in amount in comparison with normal cerebellum.

Pons, Medulla and Cord.—Almost universally the cells show a greater or less degree of degeneration, with general swelling, displacement of the nucleus, and chromatolysis. It is rare to find a cell with normal appearance. In general the cells of the anterior horns are rather more affected than those of the posterior horns. The cells of Clarke's column are similarly affected. In Weigert-Pal and Marchi preparations there appears to be slight, diffuse degeneration in all columns, least marked in the posterior. The optic chiasma when stained by the Weigert-Pal method shows but little change. Sections of the internal organs, of the thyroid, thymus, adrenals, &c., showed nothing abnormal.

PATHOLOGICAL EXAMINATION OF JESSIE B., THE FOURTH MEMBER OF THE ABOVE FAMILY.

Nothing abnormal was observed in regard to the membrane or the surface of the brain. The thoracic and abdominal viscera were normal. After hardening in formalin the pia mater was removed and the brain examined. The convolutions were well formed and nothing abnormal could be detected either on the surface, base, or in cut sections of the brain or cerebellum. The liver and spleen also appeared normal, both macroscopically and microscopically.

Microscopical examination of the nervous system was carried out by the Weigert-Pal, Marchi, Nissl, Van Gieson, and Bielschowsky methods.

Marchi Method.—Section of the cerebral cortex from the upper portion of the precentral gyrus showed a considerable amount of degeneration in the fibres of the white matter streaming down from the cells of the cortex, but very little degeneration could be seen in the grey matter. The degeneration from the precentral gyrus could be traced into the medulla and spinal cord. Sections taken from the frontal, post-central and occipital region of the cortex show comparatively little degeneration by this method. In the cerebellum similar degeneration could be seen in the fibres of the white matter passing from the grey matter of the cortex. The cells of Purkinje appeared to be diminished in number and those present stain rather darkly with the Marchi method. In the medulla there was considerable degeneration in the pyramidal tract, in the transverse fibres, and also in the intramedullary portion of the cranial nerves. In the cervical, dorsal, and lumbar region of the spinal cord there was a diffuse degeneration not only in the pyramidal tract but also in the antero-lateral tract and posterior column. There was some degeneration in the ventral roots and a few degenerated fibres could be traced to the cells of the anterior horn. The dorsal roots show but little change.

Weigert-Pal Method.—The medullated fibres of the cortex were well-stained, and the tangential fibres of the cortex appeared to be about normal. No abnormality was to be seen in the spinal cord stained by this method. The cerebellum showed some diminution of the medullated fibres. Sections of the optic chiasma showed but little change, as did also sections of the optic nerve. The eyes of this case were unfortunately not preserved.

Van Gieson Method.—No change could be detected in the vessels, either in the brain or spinal cord, by this method and no inflammatory reaction was present. The membranes of the brain and cord appeared normal.

Nissl and Van Gieson Method.—Cerebrum: Sections of the cortex taken from the upper portion of the precentral gyrus stained by the Nissl and Van Gieson methods showed very marked changes in the pyramidal and Betz cells. The cells were few in number, some were small in size, others swollen, and many of them showed an excentric nucleus and vacuolation, and diffuse staining of the chromatophilic substance which tended to arrange itself around the nucleus.

Cerebellum: The Purkinje cells were diminished in number, had lost their dendritic process, and were swollen and vacuolated. Some had central nuclei, but the cell body was poorly and diffusely stained. A layer of large granular cells was a very marked feature in the region of the Purkinje cells. The deeper granular layer was very poor in cells. Stained by the Bielschowsky method, similar changes were shown. Spinal cord: The cells of the ventral horn of the spinal cord were numerous and showed changes similar to those above described, but compared to the cells of the cerebrum and cerebellum were well preserved.

SUMMARY.

Shortly, the clinical features in this family are as follows: Out of a family of five children, born of healthy non-Jewish parents, three were affected with a progressive disease leading to dementia, blindness and paralysis—one of whom showed changes in the macular region of the eyes at a late stage of the disease. The failure of vision occurred before the macular change developed.

The children were healthy at birth, and developed in a normal manner till the age of $3\frac{1}{2}$. Epileptic fits occurred, and they then began to degenerate mentally. They became noisy, dirty in habits, and developed a spastic condition of the limbs. Death ensued in one child at the age of 8, in the second at the age of 4, and in the third at the age of 6.

In two a post-mortem has been performed. In one case no change was visible in the nervous system on macroscopical examination, in the other only slight atrophy, but on microscopical examination diffuse degenerative changes affecting the ganglion cells were visible in the cerebrum, cerebellum, spinal cord and retina similar to those described in the Waren Tay-Sachs disease.

The Wassermann reaction of the blood and cerebrospinal fluid was negative in both cases, and no change in the brain or membranes was found suggesting congenital syphilis.

CONCLUSIONS.

It is clear from a consideration of the above cases and their pathology that there is a group of cases similar to the Waren Tay-Sachs family amaurotic idiocy, but occurring at a later age and not race selective. The disease is characterised by progressive blindness, pro-

gressive paralysis, and progressive dementia; but the changes at the macula are not always present, and when present are in some cases a late manifestation, in others an early manifestation of the disease.

The later in life the disease appears the less acute seems to be the course, and in those cases in which the visual defect manifests itself during or after the second decade of life there appears to be little or no liability for the central nervous system to be affected. The parallelism of the clinical symptoms with the Waren Tay-Sachs cases is clear. The agreement lies in (1) the familial character; (2) the absence of syphilis; (3) the clinical symptoms and course. The difference lies in (1) the absence of race proclivity; (2) the absence of the characteristic macular change; (3) the difference of age.

What the nature of the poison may be which is so selective in its mode of action and tends to affect several members of a childship is a problem yet to be solved, for neither the exhaustion theory of Edinger nor the abiotrophy theory of Gowers helps to throw light on the condition.

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DISCUSSION.

Mr. J. HERBERT PARSONS said he had examined the eyes of two cases of amaurotic family idiocy some years ago; the brains were examined, and reported upon in *Brain* by Dr. Gordon Holmes, and the clinical condition by Dr. Poynton. He agreed with what Mr. Mayou now said as to the condition of the fundus. He (the speaker) stated that there was a very extensive condition of œdema in the neighbourhood of the macula, with vacuolation of the cells and cystic formation in the retina. He did not think his own particular case was fixed as well as was Mr. Mayou's specimen, but he was interested in Mr. Mayou's confirmation, because there was an opinion prevalent that the condition was not œdema, but a coagulation necrosis; he believed Mr. Coats, after examining a case, arrived at this conclusion. The view was that there was not a hole in the macula, and that the cystic formation and the œdema were to a great extent a post-mortem occurrence—an artefact. He could not reconcile the extraordinary difference in the ophthalmoscopic appearance of Dr. Batten's cases and true amaurotic family idiocy. Doubtless all the cases which Dr. Batten had described were all of one type; but he believed the literature showed that whereas amaurotic family idiocy occurring in Jews was an absolutely distinct condition—a sharply cut syndrome with an extraordinary similarity in all the cases, and always, until to-night, associated with the Jewish race—that was not so with other somewhat similar states, and in the present state of knowledge it was not, he thought, advisable to mix them up together. Moreover, the cases which did not occur in Jews were not very typical. Cases of cerebral degeneration with macular changes had been confused in a hopeless manner. He asked whether the consistency of the brain in these cases described by Dr. Batten was as characteristic as that in cases of amaurotic family idiocy; the latter were of gutta-serena-like hardness. He asked whether the sequence of symptoms in various parts of the body affected in these delayed cases was the same as in the amaurotic family idiots. In the Jew cases the neurological syndrome was very characteristic.

Mr. TREACHER COLLINS remarked that many years ago he examined microscopically the macular region in a case of amaurotic family idiocy described by Mr. Kingdon;¹ he believed it was the first case of the kind in which the eye had been examined microscopically. The sections showed the same appearance as that which Mr. Mayou had demonstrated;² the fold in the retina was almost an exact counterpart of that shown now, with œdema in the internuclear layer. He (the speaker) did not apply a special staining for the ganglion cell, and he thought the appearance seen in the macula

¹ *Trans. Ophth. Soc.*, 1892, xii, p. 125.

² *Vide Med. Chir. Trans.*, 1897, lxxx, pl. xix.

ophthalmoscopically was due to œdema. Some years later, Dr. Ward Holden, of New York, staining some of the sections in one of these cases, especially for the ganglion cells, found they had undergone coagulative necrosis, and attributed the œdema which he (Mr. Collins) had found to post-mortem change. The specimen which he (the speaker) examined remained some little time before it was fixed, so that the conclusion might be warranted. But Mr. Mayou had fixed his specimen immediately after death, and still found the œdema. It would seem, therefore, to be still an open question as to whether the changes seen in the macula were due to œdema of the retina or to coagulative necrosis of the ganglion cells.

Dr. F. E. BATTEN in reply, said he was sorry he had not made it clear to Mr. Parsons that the changes in the nervous system in this condition were most characteristic. If a case of Waren Tay-Sachs disease were shown to a person accustomed to examining the nervous system, he would say at once it was perfectly characteristic of the condition; most pathologists would have said the case now described was typical. Therefore on the pathological side, the cases were an exact counterpart of each other. With regard to the consistency of these brains, one of the most striking features was that they were normally convoluted and unduly firm: in the Waren Tay-Sachs disease there was a definite sequence of symptoms. First, the failure of vision as the earliest manifestation; and that occurred before the macular change could be seen. Then there was impairment of mental and physical condition. In older children it was noticed that they looked with the side of the eyes, because they had lost central vision; then they lost their recently acquired faculties, such as reading and writing. Then they began to degenerate physically, as shown in defective walking and diminished power of using the hands. It was true that in the cases described as occurring in the second decade of life, apparently there was failure of vision and macular changes but without change on the mental and physical sides. When he started to investigate these late cases his idea was that he was dealing with a group distinct from the Waren Tay-Sachs cases, and it was only after examining them very carefully that he had changed his view; and he now felt convinced that there was one large group of cases of maculo-cerebral degeneration of which the Waren Tay-Sachs was the first group; another group started at the age of 3 or 4, and another at the age of 7 or 8. The disease started at about the same age in the children of the same family. One should be careful to separate these from the cases in which retinitis pigmentosa occurred in families, for he believed they were cases of a different order. He had not, however, had an opportunity of examining the central nervous system of a case of family retinitis pigmentosa. He believed the name Waren Tay-Sachs family amaurotic idiocy would be maintained in spite of the fact that "idiocy" was, however, a misleading term; these patients were not idiots, since they had normally convoluted brains at birth.

Mr. MAYOU replied that in both the cases as much care as possible was taken in regard to fixing before the examination; and he regarded the fixation as quite satisfactory. He agreed with Mr. Treacher Collins in thinking that the change in the infantile forms of amaurotic family idiocy was due to œdema, rather than to coagulation necrosis. We did not yet know what coagulation necrosis in the retina looked like with the ophthalmoscope; certainly he did not, but thought it was very doubtful if the changes in the ganglion cells alone could produce the gross changes seen with the ophthalmoscope.

Section of Ophthalmology.

March 3, 1915.

Mr. J. B. LAWFORD, F.R.C.S., Vice-President of the Section,
in the Chair.

Cyclitis with Spontaneous Subluxation of Lens, treated by Couching of Lens.

By A. C. HUDSON, F.R.C.S.

PATIENT, female, aged 56.

May 7, 1914: R., cyclitis of one month's history. Gross, freely floating vitreous opacity. Slightly increased intra-ocular tension. Vision, $< \frac{6}{60}$; L., normal. Treatment for oral sepsis prescribed.

June 22: Spontaneous dislocation of lower border of lens forwards through pupil, followed by some slight further increase of intra-ocular tension.

August 8: Admitted to hospital; R., very shallow A.C., T., +1. Vision, $\frac{3}{60}$.

August 10: Lens dislocated downwards into vitreous by means of needle curved on flat, introduced at lower angle of A.C., with concavity of blade directed backwards. (Administration of eserine before operation had given rise to acute glaucoma with abolition of A.C.)

Intra-ocular tension remained normal after operation, under use of atropine, until October 5, when there was decided increase of tension. This was at once relieved by use of eserine, and there has been no further rise. Inflammatory symptoms subsided almost immediately after operation.

August 31: Vision, with correction, $\frac{6}{18}$.

Present condition: Vision, with correction, $\frac{6}{9}$. Vitreous almost completely clear. Tension normal.

Remarks.—The case appeared extremely unfavourable for lens extraction. The astonishing improvement in the condition almost immediately after operation is difficult to explain.

Traumatic Dislocation of Lens treated by Couching and subsequent Separation of Iris Adhesion at Angle of Anterior Chamber with Cyclodialysis.

By A. C. HUDSON, F.R.C.S.

PATIENT, male, aged 63.

August 28, 1914: Blow on left eye two days previously from piece of wood. L., severe chronic blepharitis with conjunctivitis. Extensive corneal abrasion involving central part. Lens dislocated into A.C. Maximal dilatation of pupil; T., +1. Vision, hand movements.

Same date: Left lens dislocated downwards and backwards into vitreous by means of needle curved on flat, introduced at lower angle of A.C., with concavity of blade directed backwards. Pupil failed to respond to eserine, and intra-ocular tension remained high.

December 10, 1914: L., small scleral incision made about 7 mm. to outer side of outer limbus, iris repositor introduced and pushed right across A.C. into inner angle. Adhesion of iris to cornea and sclera separated by sweeping, and to some extent by backward, movements of point of repositor, a small cyclodialysis being produced at the same time in the neighbourhood of the outer angle.

Since operation the intra-ocular tension has remained usually subnormal until to-day, when it is slightly above normal, and the condition of cornea and conjunctiva has much improved. Vision, with correction, $\frac{6}{60}$.

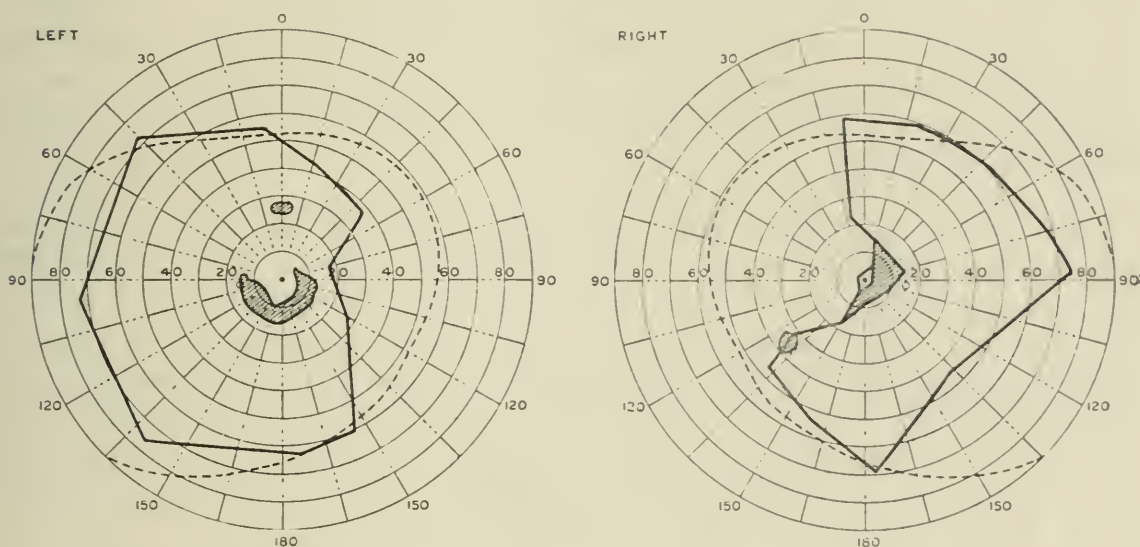
Remarks.—The case was obviously a very unfavourable one for lens extraction. The operation of iris separation at the site of a closed angle, in combination with Herbert's small flap sclerotomy, is one which the exhibitor has performed in another case with favourable results, and which he regards as worthy of further trial.

Glaucoma in a Boy, aged 17.

By M. S. MAYOU, F.R.C.S.

THE patient was seen by me five years ago. Vision $\frac{6}{6}$, with right, -2.5; left, -1.5. Always delicate; has had large tubercular glands in the neck. Since August his sight began to fail. Vision, right eye, $\frac{6}{60}$; left, $\frac{6}{12}$, -1.75 = $\frac{6}{9}$. No history of haloes. The anterior chambers are

somewhat deeper than normal, but there is no sign of any adhesion in the angles of the chambers. No K.P. signs of irido-cyclitis, the vitreous being quite clear. The optic disk in each eye presents a deep, glaucomatous cup. There is a contraction of both fields. In the right eye the nasal half of the right field is largely affected, and it passes up to the fixation point. In the left eye the nasal half is affected to a much less extent, and below the macula there is a big scotoma which is



Fields of vision.

absolute, and again even between the 20° and 30° circle there is a smaller one. The patient was trephined in the right eye on January 5, 1915, and in the left eye on January 19, 1915. He made an uneventful recovery from the trephining operations, the areas of which leaked freely subsequent to the operation; the only point of interest being that he had severe hæmorrhage from the right iris at the time of the operation, and in both eyes there were repeated small hæmorrhages into the anterior chamber subsequently (no history of hæmophilia). Since the operation, although the tension does not seem to have increased above the normal, there seems to be an increase in the size of the scotoma below the fixation point in the left eye, and it now nearly reaches the fixation point. The patient complains that coloured light obscures his view of objects.

DISCUSSION.

The CHAIRMAN said Mr. Mayou's case was one of interest, especially in view of the youth of the patient. He asked whether Mr. Mayou could tell the meeting anything about the family history. In cases in which glaucoma occurred at an abnormally early age, it was not unusual to find that it was inherited and an example of "anticipation." The youngest case of primary glaucoma he remembered having seen was that of a girl, aged 15, and in that case a family history of the disease could not be obtained. He gathered from Mr. Mayou's remarks that he regarded the scotoma in the left eye below the fixation point, which was continuous with the blind spot, as something unusual. He (the Chairman) did not think it was uncommon, in cases of glaucoma of the quiet kind, to find scotomata in that part of the field, usually within the 20° circle.

Mr. HERBERT FISHER said his impression of the case was that it was probably a congenitally imperfect angle, although it was not possible to detect the obstructing piece of iris which was at the back of the cornea and in contact with it. One knew that glaucoma was a common experience in cases of congenital aniridia, in which there was no obvious iris. The fact that the aqueous chambers were not shallow, but deep and apparently roomy, was against this being ordinary acquired glaucoma. It would be of interest to know what were the diameters of the corneæ. If they were full, that fact would favour its being a case of congenital malformation.

Mr. MAYOU replied that no other members of the family were affected. The corneal diameters had been measured, and were approximately normal.

Bullet Wound of Orbit.

By G. WINFIELD ROLL, F.R.C.S.

THE patient, shown with the healed cicatrix of a bullet wound, is a private in the Grenadier Guards, aged 30, who was wounded at the battle of the Marne.

The bullet entered on a level with the floor of the right orbit, where a notch can be felt just external to the infra-orbital nerve. The cicatrix of the wound of exit is close to the tragus of the right ear. There is no affection of hearing, but apparently the capsule of the mandible was wounded and there is slight difficulty in widely separating the teeth. The patient complained immediately of loss of vision, and for some time

the right eye appears to have been prominent from blood extravasation in the orbit. Vision: R., $\frac{6}{36}$; L., $\frac{6}{6}$. At the centre of the macula is a circular opening with well-defined edges about half the size of a papilla. The surrounding area is slightly opaque and paler in tint, with some scattered bright dots on it. The floor of the aperture is uniformly red and shows faint mottling. The remainder of the fundus is normal, and also the structure of the globe. From the direction of the wound there was obviously no direct injury to the globe, and the effect is no doubt a contusion due to the shock of the bullet's impact on surrounding parts, bony and soft tissues.

The interesting point arises as to the patient's further value as a soldier. Obviously he is of no further use in a shooting regiment unless he can adapt himself to altered conditions and fire his rifle from the left shoulder. As regards the injured globe, the prospect of improvement of vision is not good.

Case of Proptosis occurring in a Male Child, aged 6.

By G. WINFIELD ROLL, F.R.C.S.

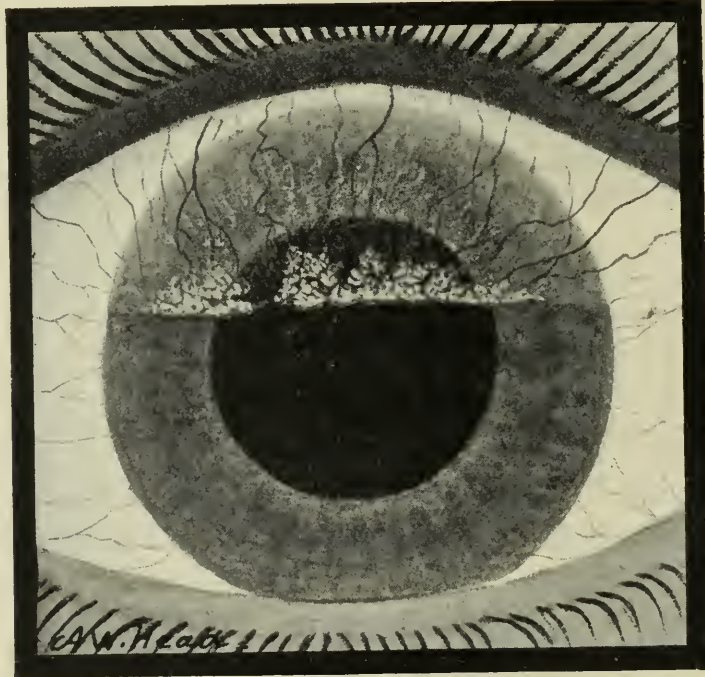
THE left eye is prominent and has been so during the last three years. The mother thinks that it was not present or noticeable in the first years of life. The ocular fundus is normal and visual acuity = $\frac{6}{6}$. Apparently the boy suffers neither pain nor inconvenience. Examination of nasopharynx and nares negative. The patient has been twice submitted to X-ray examination without result. There is a decided asymmetry of the two sides of the boy's face; the right side is rounder and better developed. The eyelids can be completely closed. There are no general symptoms to indicate Graves's disease.

Mr. HERBERT FISHER said he knew of a case of typical Graves's disease in a girl, aged 9, the daughter of a doctor. It was a typical and aggravated instance.

Vascular Keratitis with Terminal Perivascular Deposits.

By RAYNER BATTEN, M.D.

PATIENT, female, aged 15. Attended the Western Ophthalmic Hospital in January, 1915. The condition of the eyes had only been accidentally noticed the day before. There is no history of



Vascular keratitis with perivascular deposits. Right eye.

any previous eye or lid trouble. It is uncertain how long the condition had existed. Vision: R., $\frac{6}{18}$, J.1 \bar{c} + 4 cyl. $170^\circ = \frac{6}{12}$; L., $\frac{6}{18}$, J.1 \bar{c} + 4 cyl. $170^\circ = \frac{6}{12}$. In the right eye the conjunctival vessels spread on to the upper third of the cornea, resembling in their distribution a pannus and terminating in a horizontal straight line. The termination of each vessel is surrounded with a conical egg-shaped area of bright white deposit with a faint milky halo round its distal end. The deposits, following the distribution of the vessels, form triangular groups of deposits, which extend in a horizontal line across the cornea. The condition would appear to be progressive, and the

area of the cornea previously invaded is dotted with fine flecks of deposit, forming a thin white cloud. There is a similar condition in the left eye, but less marked. The corneæ do not stain with fluorescin. The deposits do not feel gritty to the touch. The conjunctival surface of the eyelids is quite smooth and there is no history or evidence of past trachoma.

DISCUSSION.

Mr. BATTEN said that the patient had + 4D. of astigmatism, and he did not think the change in the cornea could be the cause of the astigmatism; but it was possible that the spasm to correct the astigmatism might produce the vascularity seen. It had just been pointed out to him that with high magnification there were also fine white dust-like particles throughout the rest of the cornea.

The CHAIRMAN asked whether Mr. Batten could state anything about the history of the case, especially as to any previous eye or lid trouble. He did not think the conjunctivæ could be called normal, though it was true they were not follicular or granular. The upper lids were markedly thickened, and there was noticeable ptosis, more marked in the eye with the worst cornea.

Mr. ERNEST CLARKE asked what treatment Mr. Batten proposed to carry out.

Mr. BATTEN replied that the only piece of information the mother could give was that the child once had "blight" for a few days; otherwise she had had no trouble with the eyes. At present the redness and œdema of the conjunctiva was more marked than previously. He thought the amount of conjunctivitis present was due to her having so much astigmatism. There was no family history of any such condition. In reply to Mr. Ernest Clarke, he proposed to do a peritomy and cauterise.

Conical Cornea with Raynaud's Disease.

By CHARLES WRAY, F.R.C.S.

THE patient, a female, aged 26, has had trouble with her sight for six years, but never had to sit on the front benches at school. There is no swelling about the neck, and no tachycardia or any symptoms associated with disease of the thyroid gland. The patient states that her general health is good, but the hands are always bluish-red, congested, and very cold to the touch. The finger-nails are thin and

somewhat ill-developed. Contrary to rule, the cone is central and the summit occupies the centre of the cornea. Doubtless the absence of the usual semilune of comparatively sound cornea skirting the upper part of the cone explains the poor vision obtained by the correction of her refraction. The keratometer shows astigmatism 5D., axis 30, or 120 in the right eye. In the left the astigmatism appears to be 8D., axis 70, or 160. The subjective result is as follows: Right eye, vision $\frac{6}{60}$, $\bar{c} + 6$ cyl. at 30 = $\frac{6}{18}$; left eye, fingers at 3 metres, $\bar{c} + 10$ at 30 = $\frac{6}{36}$.

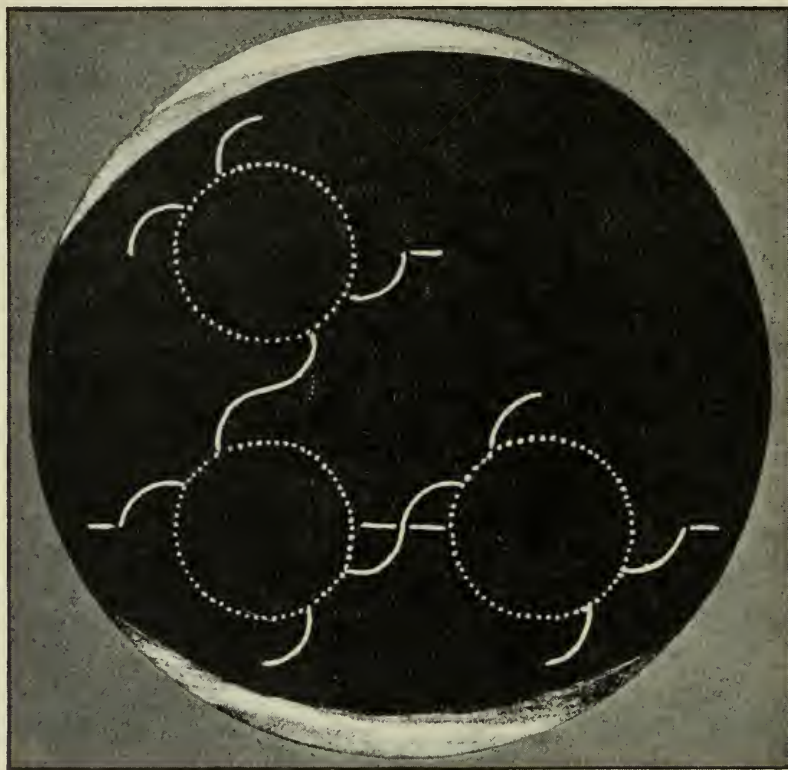


FIG. 1.

Keratometer images as in normal cornea. Normal eye.

Below are drawings of the cornea as seen when the maximum attempt has been made to measure the astigmatism by means of Sutcliffe's keratometer. Figs. 1, 2, and 3 show the final appearances in the estimation of astigmatism in the normal eye and the patient's right and left eyes respectively. It will be noted it is immeasurable in the left eye as the upper circle cannot be brought into contact with the lower.

The case will be re-exhibited after treatment.

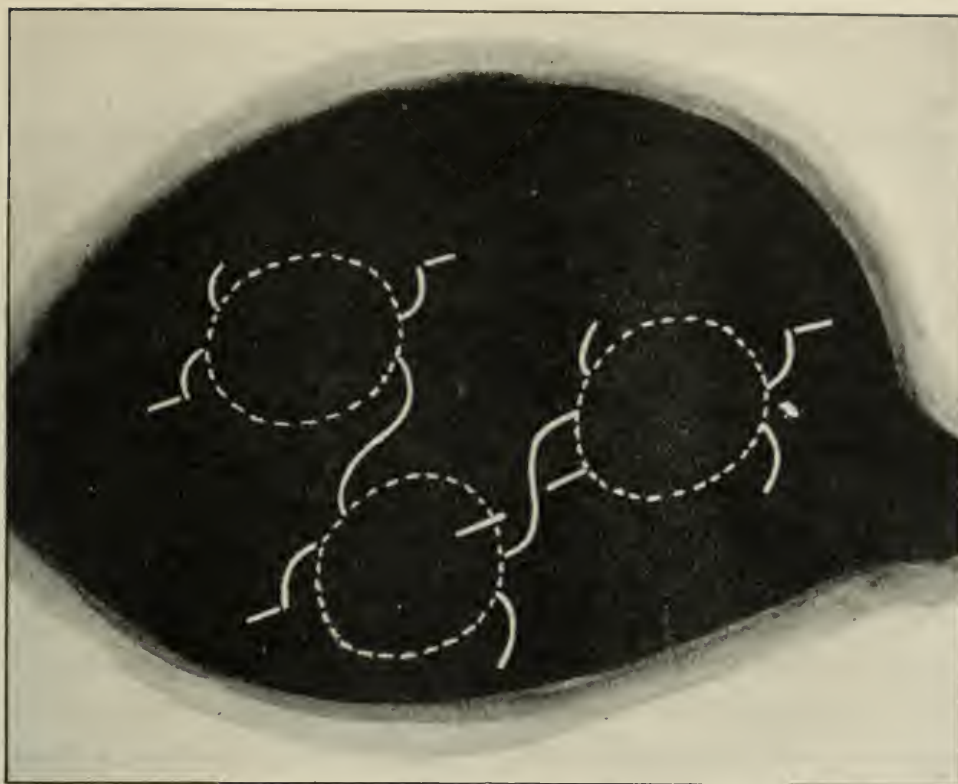


FIG.
As seen in +8D. conus astigmatism. Right eye.

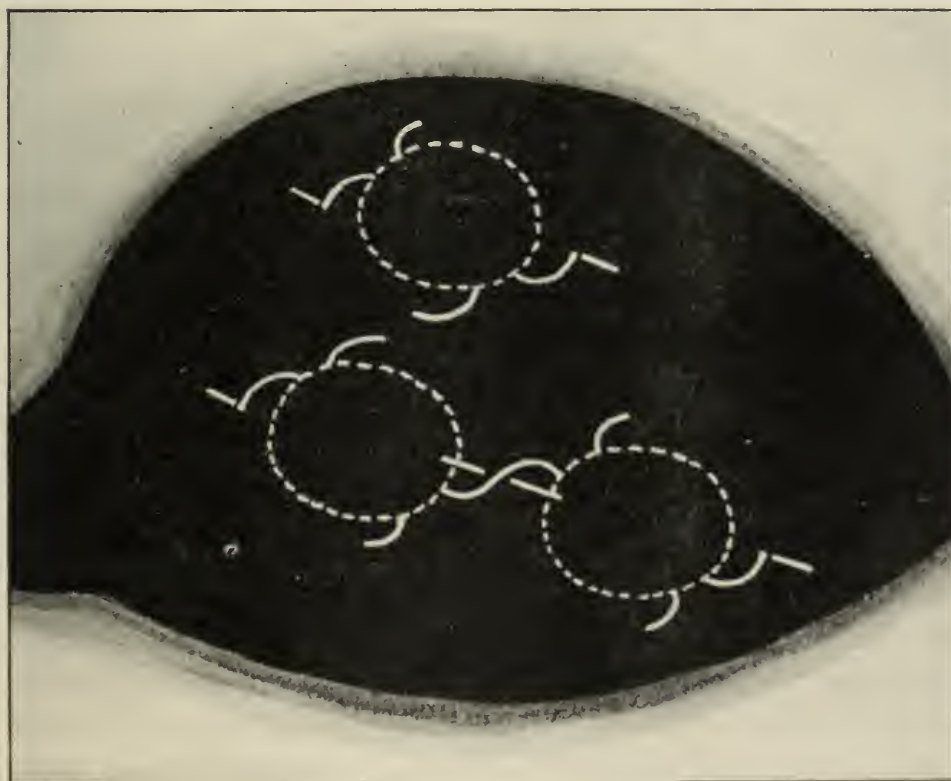


FIG. 3.
In very high grade conus astigmatism not measurable by means of the keratometer.
Left eye.

Montessori Education for Children with Defective Sight.

By LESLIE PATON, F.R.C.S.

MY attention was first called to the value of Madame Montessori's system of education and its possibilities in training children with very defective sight by the case of a small patient, aged $2\frac{1}{2}$, when I saw him first. Severe ophthalmia neonatorum had caused blindness in the left eye and very defective vision in the right eye. In July of last year when I saw him again he was aged $3\frac{1}{2}$, and I was very greatly impressed by the general intelligence of the boy and by the fact that despite his great visual disabilities he already knew his letters and was able to count small numbers. The parents ascribed this to the training he had had during the preceding six months at a Montessori school. I have since had occasion to investigate a little more fully the value of this method for the training of normal children, and though I have no more than a superficial book knowledge of it, it seems to me to have possibilities for training blind children or those with defective sight which, as ophthalmic surgeons, we should know about and, if possible, develop.

My subject-matter will be in no way new to those who have taken an interest in problems of child psychology or modern elementary education, but even in quarters where such an interest might be expected to be most active I have found the most complete ignorance on these subjects, an ignorance which I shared a few months ago. For the education of children of school age with defective sight much has been done in recent years, and only the year before last Mr. Bishop Harman read an excellent paper before the Section on methods in use.¹ But it is for younger children in the very important formative period of three to six that the Montessori methods are at present adapted. During those years the child is undergoing its most important sensory education. It is learning to use not only all the ordinary senses of sight, hearing, touch, taste, and smell, but also the muscular and stereognostic senses, and to associate the percepts arising from these various senses and to co-ordinate its movements. Froebel stood almost alone in the attempt

¹ *Proc. Roy. Soc. Med.*, 1913, vi, pp. 146-163.

to systematise education during this important period of a child's life, but in many quarters it was and still is regarded as sufficient to allow the normal practical contact with the surrounding and expanding world of the child's life to give it the experience necessary for its development. Only in cases where the child remains in an obviously defective condition after reaching normal school age were attempts made to adopt systematic methods of dealing with these sensory defects. The most important work in this line was done by Seguin, who published his "*Traitement Moral, Hygiène et Education des Idiots*" in Paris in 1846. This work, though it avowedly deals with the treatment of defective children, is based on such sound psychological observation of child life and development that to the present day it forms the foundations on which all child educators must build. Madame Montessori herself acknowledges in the fullest fashion all that she owes to Seguin and to her early experience in dealing with defective children. Indeed, it was mainly the fact that defectives could be brought to such a level of accomplishment that they could compete on equal terms with normal children of the same age which induced Madame Montessori to realise that the methods in use for normal children must be in some way lacking, and determined her to take up the question of applying Seguin's methods to the teaching of normals. She gave up her medical work entirely and devoted seven years to the study of pedagogy and psychology, especially the psychology of the child, and it was only in 1907 that the first school under the Dottressa's guidance was opened in Rome. These intervening eight years have witnessed a great development and extension of the method, so much so that even in this conservative country several classes have already been started. I know of three schools in London, a very flourishing small school at Runton, and one in Birmingham, a class in a village school in the New Forest, and large classes in Elementary Schools in York and in Sunderland. The main difficulty in the way of the extension of the system, apart from the inertia of the teaching profession and the public or the parents, is the difficulty of finding the right conductor. I avoid the use of the term teacher because the essence of the method is to allow the natural development of the child to take place with the most scrupulous avoidance of any attempt to force it into a common mould. It requires a patience worthy of Job himself to avoid interfering when a child is seen doing an exercise wrongly time after time; but the reward comes in the intense satisfaction displayed by the child when at last he

succeeds unaided in doing it rightly. Much of the training is of such a character that the child can be left to work out the correct method by himself, and in some quarters this feature has given rise to a false impression that the children are allowed to do as they please. This is quite untrue if it is meant to imply an absence of discipline; but in the sense that the child is encouraged to develop his natural healthy aptitudes fully and freely, it is true. A child who is tired of one particular exercise or who finds it beyond his capacity will gain no benefit by being kept at that exercise. If he is interested in it no outside enticement will take his mind off it until he has achieved it. This is one of the important points in which it differs from the kindergarten system, where it is supposed that it tires a child's brain to keep it more than a limited space of time at one exercise. The result is that as soon as the child becomes thoroughly interested in one thing his mind is distracted into another quite different groove; as an intelligent small boy remarked, "The lesson was all interruptions." I feel sure this is based on a wrong idea of child psychology, and that so long as the child is interested there is no chance of brain-fag. Constantly changing the channel in which the child's mind is running is much more likely to tire it out. A child left to itself possesses a power of concentration on the work or play on hand which might serve as an example to many of its elders. Surely the Montessori system, which tends to conserve and develop this power, is preferable to one which dissipates it. Within the limits of a short paper I can only deal briefly with the material basis of the system, but I hope it may be sufficient to let the Section realise how easily it could be adapted for the use of children whose sight is lost or defective. Indeed, even with normal children many if not most of the exercises are done frequently blindfolded to educate the tactile, muscular, and stereognostic senses. And if it is important to train these senses in normal children, how much more important is it to do so in the child who is deprived of that sense which for most of us is the main one to which our educative processes have been adapted. Before I go further I think it may interest members to see what kind of result can be reached in education in the ordinary limited sense of the word. I ask you to look at a sample of writing by a child aged 5. A sense of shame alone prevents me from putting up for comparison a sample of writing by one who only painfully acquired the art by copying pot-hooks and copy-book phrases. Vision in the Montessori system plays a very small part in learning to write.

The art is acquired almost casually as a by-product in training the tactile and muscular senses.

A child aged 3 is first started on what may be called exercises of practical life—buttoning, lacing, fastening hooks and eyes, and tying bows. I have seen a normally restless and over-active child sit quietly for an hour over one or other of these frames, making many mistakes, but realising them for herself, and not in any way relishing the interference of the intrusive elder who wishes to show her how to do it, and ultimately triumphing in doing it correctly unaided. This is the first exercise in muscular co-ordination. The second series is devoted to the tactile sense. It consists of boards covered with strips of sandpaper and fabrics of different texture and surface, which the child learns to differentiate blindfolded. Then come exercises in the discrimination of simple sounds gradually leading up to musical sounds and the development of a sense of rhythm. With this is associated the teaching of the art of moving quietly and gracefully, and, in a later stage, dancing.

Perception of weight is taught by means of blocks of wood of the same size, but of varying density.

The next series of exercises introduces the child to the idea of dimension. Three series of inset blocks, which vary (*a*) in length only, (*b*) in breadth or diameter only, and (*c*) in length and breadth. Starting with one set at a time, the child learns to fit them all into their proper places, until finally it can do the exercise with all the blocks mixed together. This exercise is done both visually and tactually, and, ultimately, as tactile discrimination develops, a child will fit every block in without mistake when blindfolded. In this series the apparatus is self-corrective, and the child can be left absolutely to itself to play with the insets. The idea of dimension is carried further by the "tower," cubes varying in three dimensions, the broad stair, bricks varying in two dimensions, and the long stair, where only one dimension varies. This also serves to introduce later on the idea of number. The child, of course, is told the names of the different dimensions, and learns to appreciate length, breadth, and height.

The next teaching material introduces the idea of form. It consists of a large series of wooden frames with geometric insets of various kinds. These are used first of all visually, and then blindfolded, and the child fits the form into the frame by tactile muscular sense. From the point of view of education in the narrower sense of the word this forms one of the most important steps in the process of learning to

write. The child in a very short time gains such a control of the tactile muscular sense that simply by feeling the outer edge of the insets and the inner edge of the frames it is able to fit in any one of these figures from the simplest to the most complicated. At the same time it is taught the names of the different figures, and though it may seem absurd to talk of a child, aged 4, knowing anything of geometry, it is actually acquiring ideas of shape which are the basis of all geometrical teaching, and this is carried further by a series of cards reproducing these shapes, first in solid colour, then in thick outline, and then in thin outline, so that the child fitting the insets over these is gradually carried over mentally from the concrete form to the abstract idea of spatial relations limited by lines.

I am afraid the training of the colour-sense will hardly be applicable to blind children, so I pass on to the next material, which consists of metal insets similar to the wooden ones. With these the child commences to use pencils and coloured chinks, forming the outlines on cards and filling them in with colours. At first the colours may transgress the bounds of the outlines wildly, but in an astonishingly short space of time muscular control is gained, and the figure is filled in with strong, firm lines. This exercise would probably have to be modified for children with defective sight, and, instead of outlining the inset, the outer frame would have to be made of metal, and, after outlining the figure, the child would use the frame as a control for filling in the figure. It would thus gain in a similar way power of muscular control over a pencil.

The first alphabetic material consists of script letters cut out in sandpaper and mounted on cards. These the child learns more by touch than by vision. The card is held in the left hand, and the right index-finger is passed lightly over the sandpaper from left to right, and the name of the letter is repeated. For normal children the vowels are printed on blue and the consonants on pink. With blind children different roughnesses of sandpaper or some difference in surface texture could easily be devised for differentiating purposes. As the child is learning these letters by tactile muscular sense, it is at the same time practising the movements of muscular co-ordination, which are necessary for the formation of the letters, and as it has gained already the power of control of a pencil or chalk, it suddenly realises its power of combining these two acquirements, and without any further instruction writing is an accomplished fact in that child's training.

Mr. Holmes, in his report to the Board of Education, gives six

weeks as a very ordinary time from the beginning of the training till the writing of actual letters. With older children, aged 6 or more, this may be reduced to a month, and so excellent is the control of muscular movement that from the very first the writing of Montessori children compares favourably with the writing of third and fourth standard children, and, as I have already said, compares more than favourably with the writing of some adults whom I could mention.

When we come to reading we are faced in this country with a difficulty that is not met with in Italy. Italian is probably the most purely phonetic language in Europe, and the child who has learned the sound of the various letters has only to name these successively, with gradually increasing rapidity, until he recognises that they form a word which he knows. English is probably the least phonetic language in Europe, and, at least to begin with, great care must be exercised in selecting words which are phonetic. The letters for teaching the formation of words are cut out of the cardboard and unmounted. The child has meantime learned all their names, both visually and tactually, but you may still see them, if they are a little doubtful, passing their fingers rapidly over the letter. It would almost seem as if the tactile impression conveyed a more vivid impression to their intelligence than the visual.

In teaching numeration and arithmetic, we go back first to the long stair which increases by lengths of one decimetre from one to ten. The names and shapes of the numbers are learned by means of sandpaper figures like the letters, and counting and addition and subtraction are learned by means of the counting boxes and sticks, and for numbers over ten by wooden frames with printed numbers.

This material substratum of which I have been speaking is only a part, I might almost say a small part, of the method. The whole method itself is of no avail if the principle underlying it is not fully appreciated. Of course it is revolutionary, a reversal of previously accepted notions of elementary education. It is the free development by the child itself of all its inherent capacities, as opposed to the inculcation from an outside source of dogmata which the child may or may not accept willingly or sullenly, but which as they are usually enforced do not easily become a part of the child's intellectual concepts. Everything that he acquires by the Montessori method he acquires for himself. It is part of his own intellectual being built up from his own sensory percepts. It is he himself who has learned to observe and to associate the resulting ideas, and his knowledge grows from natural

roots, and is not a forced graft from an outside source, and lest it might be thought that there is anything forced or fatiguing about the almost uncanny rapidity of progress, I may definitely say that these children are all the time quite unconscious that they are doing anything more than playing at a most absorbing game, and the weeping is very bitter if for any reason they are kept away from this game.

Possibly the most important feature in the whole method is the development of the sense of self-discipline in the child and its unconscious realisation of its social duty to other children. Mr. Holmes, late Chief Inspector of Elementary Education, speaking of this factor, has remarked how speedily even the most turbulent and unruly children become aware of their duty of self-control or have it enforced on them by the example of the community. By no other method can there be so full a development of the sense of individual freedom with self-discipline and the sense of duty as a member of a social comity. And he went on to say that Montessori-trained children, when they came under the more restrictive discipline of ordinary school-life, instead of proving less amenable, are more easily controlled than the ordinary child.

For visually defective children some slight modifications would be necessary in some parts of the apparatus. In learning numeration the long stair, instead of being made in alternate colours, blue and red, would have to be made with alternating rough and smooth surfaces. Sandpaper figures would have to be used in the counting-boxes. Frames and blocked sandpaper letters would have to be used in forming words, but so efficiently are the tactile muscular and stereognostic senses developed that I believe it would be possible to get a blind child to write on ordinary paper, keeping a moderately straight line, without the use of any of the mechanical devices which most of them have now to use. Helen Keller is a revelation of the possibilities of continuous and assiduous care in training where blindness and deafness were combined. For those less affected I believe Madame Montessori's system has possibilities capable of producing results almost as miraculous, and the purpose of my paper is simply to call the attention of the Section to it in the hope that it may be of use to members who may at any time be called on to give advice as to what is to be done in regard to the training of children who are blind or whose sight is defective.

The Education of Children with Defective Vision.

By N. BISHOP HARMAN, F.R.C.S.

IN this paper it is proposed to present a critical review of the Montessori system of education, particularly in reference to its possibilities in the education of children with defective vision, for which purpose it has been recommended as ideal; and further to draw a comparison between this system and that of "Myope Classes" which originated in this country for this same purpose, and which, after an experience of seven years, may be taken in order to show what good points they possess. A detailed account of the present development of the myopes classes will be added.

THE MONTESSORI SYSTEM.

In considering any scheme of education it is well to understand how it came into existence, and in particular to recognise the local circumstances that supplied a reasonable ground for its adoption. Next, it is well to examine the "scientific basis" on which it is founded, if indeed there be a claim for such basis. Lastly, it is well to inquire how far the local conditions of the place of its birth find a parallel in other places to which it is proposed to transplant it in its original or modified form.

The Montessori scheme of education is of Italian origin. It was first devised as a mode of instruction for mentally defective children. Its one special feature, when considered in relation to the existing modes of education then in vogue in Italy, was that a real attempt was made to allow for the personality of the child. The scheme did not aim at a uniform drilling of the minds of the children, but it aimed at awakening whatever of good there might be in the particular child before the teacher. As practised by Madame Montessori, the scheme quickly justified itself, for it was far in advance of anything known in the place of its birth. In its revolt against a cast-iron uniformity it went back to Nature and accordingly was bound to succeed, and it did succeed.

Subsequently the scheme was applied to the teaching of normal children, and in so far as it followed the natural lines of allowing for

the personality of the child it was an advance on any method that ignored this vital point.

Later the authors of the scheme developed an elaborate scientific argument, asserting the basis upon which the scheme was founded. That argument is to be found in a weighty volume, "Pedagogical Anthropology."¹

It is conceded at the outset that the scheme is a distinct advance in method upon any educational modes existing in the place of its origin. But when it is proposed to transplant such a scheme to another habitat it is well first to ask if the conditions existing in the proposed new sphere of exploitation make it desirable that it should come into use there, not merely as a new and possibly useful assistant or even competitor, but as a destructive rival of existing methods. The introduction of the scheme into England has been strongly advocated, and as vigorously opposed. And, for my part, I am in opposition to its introduction, for this reason—namely, that the grounds that promised the success of the scheme in Italy do not exist in this country; indeed, I very much doubt if they ever have existed here.

Uniformity is a thing that is totally foreign to the mental habit of the inhabitants of our islands. We are individualists from our youth up. And so strong is the recognition of this fact, that the schoolmasters do not attempt to assert any iron sway of uniformity in our public school system of education. It may be said with much truth that the chief educators of our country are the children themselves; in the friction of their several individualities there is an influence for good, better than all the schemes of the professed educationalists. Under such conditions it matters little what may be the theory of our education, the practice remains correct, and that is the chief thing.

The same state of affairs exists even under the apparently set forms of education in our elementary schools, all of which have to conform to a "Code" set by a Government board. Despite the Code there is the same play of individuality in the elementary school, the Council schools, or old "Board schools" of a more familiar nomenclature. And this recognition of personality is the more pronounced, so far as the classroom is concerned, in the infant departments, where alone any comparison with a scheme such as the Montessori could be made. The fact is, that all the premises advanced by Madame Montessori have been

¹ "Pedagogical Anthropology," by Marion Montessori (translated by Frederic Taber Cooper), Lond., 1913.

recognised in this country for generations, and have been acted upon to such an extent that no one has ever thought of talking about them.

To make an exact comparison with the Montessori scheme we should confine our judgment to our schools for mentally defectives. In this branch of education I dare assert there is no better equipped country in the world than England. In England alone there are 183 certified elementary schools for these children, with an average number of 13,110 children on the register; and from my knowledge of the London schools I would assert that it would be difficult to better the modes of training therein adopted. The real reason for the success of these schools lies in the nature of our national character. Not only is there a respect for the individuality of the child with the corresponding desire to cultivate it, but also a subtle sense of sympathy with the weak and disabled, the possession and exercise of which can alone enable the teacher to tolerate a constant association with human beings who are below the level of normal mentality and character. The Montessori system in its one good feature, the feature that has made it a success in the country of its origin, has been anticipated long ago in this country, and to attempt to import it into this country is truly a carrying of coals to Newcastle.

Secondly, it is worth while considering the "scientific basis" for this scheme as propounded in that weighty volume "Pedagogical Anthropology." Madame Montessori avows herself a disciple of Cesaro Lombroso. What he claimed to have done for criminal anthropology the author of this book seeks to do for pedagogy. She writes: "The credit rests with Italy for having rescued anthropology from a sort of Olympus, and led it by new paths to the performance of an eminent and practical service." Until these new paths were discovered "anthropology failed to raise itself from the status of a pure and aristocratic, in other words a superfluous, science, a status that prevented it from ranking among the sciences of primary importance." Lombroso's work has been subject to the most extensive and explosive criticism, and there is little left of the elegant superstructure of his criminal anthropology save perhaps in the lines of the *feuilleton* of the halfpenny daily papers. So recent a piece of work as that entitled "The English Convict: a Statistical Study," by Dr. Charles Goring, proves conclusively from the life-histories of 3,000 convicts that there is no such thing as a predestined type of humanity "born to do evil," and that "there is no definite line of demarcation, no absolute difference in nature, as opposed to degree, between the human beings who are and those who are not criminal."

Madame Montessori's scheme of pedagogy is confessedly based on anthropology of the Lombroso type. She believes that certain marks are definitions of character, but her indications are inconsistent. In one part of the book the glory of the face is made the prime index of character; in another the hand is cited as the one and only indication of capacity—"we can judge from the hand whether a man is fitted for work or not." Putting aside the inconsistency, such claims as these when applied to educational methods are destructive of all sense of impartiality in the teachers. It is hard enough for the best balanced teacher to give due attention to the plain lumpish child when there is a fascinatingly pretty, possibly mischievously roguish, child in the next seat; but to teach the teacher that the lumpish child is a lump, and therefore unworthy of the same attention as the rogue, would be disastrous, for we are not all what we promise to be.

Lastly, I wish to consider this Montessori scheme as regards its suitability for the training of children with defective eyesight. At the outset there is this difficulty: Most of the children whose eyes are found so defective as to necessitate their receiving special educational attention are not discovered until they have reached such an age that they are no longer fit subjects for such an elementary scheme of teaching. This Montessori scheme deals only with the beginnings, and the affected children have for the most part passed that period when discovered. But even supposing that there should be found a number of children of fit age and state of development, would the system be of advantage to them? If we take the claims of the scheme as set forth by its most enthusiastic supporters, the answer is emphatically—No. One of the chief and most reiterated claims made on behalf of the scheme is that children taught by this system learn to read, as it were unconsciously, fully two years before children taught by ordinary methods. Such an assertion, taking it at its face value, is quite enough to warrant us as ophthalmic surgeons withdrawing our support from the scheme. We do not want children to read two years earlier than they ordinarily do. To read early is no gain, in fact it means an ultimate loss. The child who acquires an early facility with printed books learns to forsake the true methods of learning; it ceases to pester its seniors with questions that bubble up from its growing mind, it no longer worries into the why and wherefore of this and that object that it finds in the garden and field, for its little mind is awl with the first narcotic of its life, it is learning to forget the real world in a sham fantasy of the book world. And on the physical side it is getting round-shouldered from much crouching over

print, and withal possibly straining eyes that were never meant to do this kind of thing. Educationally there is no gain in early reading, rather the reverse. Physically, early reading is a habit to be banned. A scheme that boasts this as a great merit, even if it be only in the mouths of injudicious supporters, is to be looked at askance.

It is said that the method of contact with the subject to be learned is the chief good of the scheme. Things are handled and touched, and not looked at. There is nothing new in this. There is not one of us who did not first learn to live by touch and not by sight, the baby at the breast is the ever recurring proof of this, but we soon find that of all our senses our eyes are those that give us the best and most ready appreciation of things. There are those who have to learn by touch—the blind have only this poor resource for their reading and writing. And there is no one who has seen the best of these readers but must agree in the inferiority of touch as compared with sight. If this be so with the expert blind, how much more when we consider the first hesitating gropings of the little blind child.

To suggest that we should supplant the use of the sense of vision by that of touch as the chief of our educational methods is to propose a retrogression of the most pronounced character. It would be followed to its logical conclusion if it were proposed to do away with our printed books and use Braille only. The *reductio ad absurdum* can be found in that striking story of H. G. Wells, "The Valley of the Blind."

THE MYOPE CLASS.

Special educational methods are necessary for children with defective sight, and there are methods that have been established in this country and carried on with every success. These classes are known as "Myope classes," because the greater number of children have defective eyes from this cause. In 1913 I was permitted to give an account of these classes to this Section of the Society. In London there are now five of these classes working in connexion with the public elementary schools. Similar classes have been formed in Bolton, Birmingham, Leicester, Leeds, Oldham, Stoke-on-Trent, Nottingham, Exeter, Brighton, Bristol, Sheffield, and Liverpool. Besides, classes have been established in Scotland, and in not a few cities of the United States. To judge from the published reports from the States the myope class meets as real a need there as it does here, and its exploitation proves as successful.

The myope class is in its basis a very simple and natural affair. It does not claim any extraordinary virtue as an educational revolution; but it is claimed for it that it fits a definite situation, and further that it is attractive to teachers and children alike, even though it demands greater skill in the former and more alertness from the latter.

This scheme of myope classes has been in working for seven years; it is elastic enough to meet the necessities both of the young children and of the older children, from the ages of 5 to 14, the only ages for which special forms of educational treatment are required. Its principles can be adapted to the individual needs of children attending ordinary schools, both public and elementary, and not a few short-sighted children have been admitted to well-known public schools for girls and boys and therein have followed satisfactorily their studies with such modifications in their particular interest as this myope scheme has suggested.

In the following pages there will be given an account of the present manner of working these classes in connexion with the public elementary schools; it is in this connexion that the greatest use has been made of the method, for the number of children with defective vision, of such a grade to prevent their taking safe advantage of the ordinary school curriculum, but not such as to necessitate their attendance at a school for the blind, is sufficiently large to afford ample material with which to put the scheme to the test.

Since the last account of these classes was given¹ there has been a development of the work of the classes along the lines originally drafted. The development has been particularly marked by the correlation of the teaching given in the special classrooms with that taken by the children in association with the scholars of the normal elementary schools; and further in handicraft work. The curriculum falls into three parts: (1) Oral teaching, taken for the most part in the normal schools; (2) literary work taken in the special class; (3) handicraft work also carried out in the special class. For the oral teaching the children are taken over to the associated normal school and drafted into those classes for which their attainment provides. They sit in the front row and engage in the ordinary work of the class, except that when any reading or writing work is to be done in connexion with the course of study they take no part therein. Similarly they join with the normal children for singing, drill, and games (such

¹ *Proc. Roy. Soc. Med.*, 1913, vi (Ophth. Sect.), pp. 146-163.

of them as may safely do so). Both in the normal school and in the special class much is made of action songs and musical exercises. Every effort is made to enlist the sympathies of the teachers of the normal schools with the work of these children, so that though occasional scholars they shall be regarded as part of the ordinary school work. And experience proves that these arrangements can be carried through regularly without interference with the proper work and discipline of the normal school. The children, on the other hand, learn to regard themselves as definitely associated with their normal comrades, to their great advantage both in the immediate work of their training and for the future when they leave school. So soon as one of these oral lessons is completed the myope children return to their own classroom; there the teachers of the special classes proceed to develop the lessons given in the normal schools. The work becomes in this sense somewhat of the nature of what is known as "preparation" in the public school curriculum. The lesson taken is written out from memory on the blackboard with which each child is provided, catechism is given by the teachers of the myope class who are familiar with the order of lessons given in the normal school, so that there is a very real fixation of the lesson in the minds of the children. There is further literary work given in the myope class of the kind that is usually written on paper in the normal school, but in these classes all this work is done on the blackboard, or on large printed sheets which the senior pupils in the classes prepare as part of their work. The manner of printing these sheets is as follows: Large sheets of white paper are hung up on the walls or blackboards of the room, and on them is printed by the use of rubber-faced type of 2 in. and 1 in. square selected extracts from well-known literature of the kind that can be put to educational use, and also extracts from lessons in geography, history, and the like. Already the myope classes have collected in this fashion quite a library of sheets which are in constant use by both the junior and senior children. The work of printing these sheets is greatly liked by the children, indeed it has become a prize task for the best of the elder children.

Arithmetic is practised on the blackboards, teachers and children working together on the wall blackboards and on the individual boards. A great point is made in the development of mental arithmetic, so as to familiarise the mind with ideas of figures without the adjuvant written symbols; many of the children get quite expert in the practice; this is an added safeguard against the excessive use of the eyes in

near work. Further, arithmetic is associated with practical work in handicraft.

Handicraft.—The extension of this branch of the work has been given particular attention. The provision of suitable work is by no means a simple matter. The limitations necessarily introduced by the fact that the eyes must not be used for any length of time in the stooping posture, and that the work must not be so fine as to require close application, prevent the use of some of the best, most practicable, and most educative forms of handiwork that can be exploited for school children. There is no better handiwork for boys than carpentry. It is of great interest to them; they make things that appeal to them from their form and by their utility. The task demands precision and care, and therefore is an admirable training in method and exactitude. The work grows under their hands and there are outward and visible signs of industry. The boy who puts energy into his work knows that there is fruit in his labour, for he sees it before him, and it is equally evident to him that his work has been done either well and soundly, or in a rough and slovenly fashion. But carpentry is not good for highly myopic eyes. The necessary drawing of plans, the taking of measurements, and the use of rules and scales, mean too much close work. Still more prohibitive is the posture necessary for the effective use of most of the tools employed in the work. Boys are not vigorous of muscle in their upper limbs, and this lack has to be made up by the use of the weight of the shoulders. Sawing, planing, cutting with the chisel, and the like, require muscular force, and the use of the shoulders tends to develop a stooping habit. At the same time the muscular effort is intensified by a closed larynx and forced contraction of the abdominal muscles and diaphragm, so that the very conditions of strain are induced that are most inimical to the fragile eyes of the short-sighted. These facts show how difficult it is to provide a suitable scheme of handicraft for these children.

After consideration and experiment the following scheme of work for various grades of boys and girls has been drawn up. Since boys and girls are educated together in the classes, for the most part they take the same kinds of work, and there is no special difficulty arising out of the arrangement, for the teaching is much more individual in the small classes of the myopes than can be provided in the larger classes of the normal school.

Classification.—For general convenience the children can be considered in three groups, according to age and attainment: (I) Standards

1 and 2, for those aged 7 to 10. (II) Standards 3 to 6, for those aged 10 to 13. (III) Standard 7, for those aged 13 and 14. It will be understood that there is considerable interchange in the grouping as individual children excel in one or other forms of work.

Handicraft is of value in two directions. The first and most obvious is that of the training of the "hand and eye" in the performance of useful work. That is the purely utilitarian aspect of the work, and it is the one that becomes the more evident in the higher standards. The second line of value is one of association; when some particular form of work is carried out it is possible to attach lessons in history, arithmetic, and language. This association is the more easily developed with the work of the younger children, and it is of particular value to them. For brevity's sake the first line of value will be described as the utilitarian, and the second as the expressional, since the children are taught to express some idea in concrete form.

GROUP I—STANDARDS 1 TO 2; AGES 7 TO 9.

(A) *Bead Work*.—Stringing large beads is one of the earliest pieces of work to be taught; later simple designs are worked, and finally fancy blinds for windows made. Besides the actual practice of the work there are attached lessons in arithmetic. Children readily learn to thread beads with very little examination by the eyes, provided the beads are of a sufficient size.

(B) *Paper Folding, Cutting, and Modelling*.—In this work stiff paper is used, the children are taught to fold it into certain lines, then to cut out various squares, and finally to set up the remaining portion into the form of some common object. The folding is an aid to the teaching of arithmetic, so is the cutting, and finally the object itself is a peg on which to attach an "object lesson"—e.g., a piece of brown paper 12 in. square is given to each child. (1) The edges are measured and compared. The sum of the measurements of the edges is found. The inches are then taken as feet, and the cost of binding the several edges found. The same for the measure in yards. Then the inches are taken as miles and the cost of travelling such a distance calculated. (2) The children fold bottom edge to top edge and compare sides (halves). (3) They fold into quarters and measure new distance round quarter area. (4) They fold into eighths and repeat exercises. (5) Into sixteenths. (6) Calculate cost of paper of several areas got by folding. (7) Bend over the folded paper, tuck in sides, gum them along and a model barn is produced. (8) The

children find the length, breadth, height, and distance from one side over the roof to the other. A great number of simple articles are made in this fashion, and with each is associated an "object lesson"; these lessons are therefore truly expressional, some idea is worked out in each. The following is a list of objects made by one of the classes: Trays of various shapes for trinkets, buttons, pens, silkworms; cases for pocket combs, button-hooks, needles, hatpins, cards, stamps, and spectacles; frames for pictures and photographs of various shapes and folding; boxes, with lids, and sliding; hanging spillholders, triangular, square, hexagonal, and wall tidies for hair, &c.; purse; doll's house, scissors sheath, book-case, blotter, fire screen, plant pot holders. Paper weaving, for the purpose of making small mats, and book-marks were formerly practised, but this has been stopped. Too much close attention of the eyes was required, and there was no expressional value in the work.

(C) *Modelling in Clay and Plasticine*.—This is a very favourite form of work with all children, and it is capable of unlimited expansion, both as a simple handicraft practice and in expressional work. Further, it is admirably adapted for the cultivation of the individual powers of the children. No eyestrain is involved, for the mass of material with which the work is done is large; there is no desire to bring it close to the eyes, for the size of the detail will be determined by the sight of the child, so stooping is not fostered. It is possible to associate this work with lessons in arithmetic and general history.

(D) *Toy Making*.—With the aid of such simple materials as old matchboxes, corks, sticks, and wool, a large variety of toys are made. In this fashion the children are able to produce in a material of somewhat more stable character the work of modelling. The toys are kept by the little ones and taken home, to the interest of the parents. Use is also made of the different forms of prepared wooden strips for the building up of toys, such as the "interlocking building blocks"; these are of value in dull and cold weather, with them models can be built up easily when the light is poor and hands are cold.

(E) *Drawing*.—With all the foregoing work there is associated drawing on the blackboards, or on large paper sheets in chalks. This drawing is systematic with a view to the introduction of design, and is preparatory to some of the modelling tasks, and more especially introductory to the bent iron work of the succeeding standards. Free drawing on the boards is encouraged, and some of the children develop considerable skill in picture production. In one of the schools the influence of the teachers in this direction is marked by the high grade

of really artistic work done by the children. All this work is done on a large scale, an ordinary drawing taking a space of 20 in. by 30 in., that is the full range of the child's arm. No small scale drawing such as is common in the normal schools is permitted. The children readily fall in with the habit of drawing in this fashion, and the work is always done standing.

(F) *Other Handicraft Work of a purely Utilitarian Character.*—Knitting is begun. In this care is taken to foster the habit of working without looking at the work; if a child shows no ability to do this then the work is stopped. The work done includes plain knitting, casting on and off, the making of dusters, kettle-holders, baby's hats, reins, whips, and rattles. String bag making is begun, also cane weaving, and raffia winding in the making of articles such as frames, mats, napkin rings, &c. Silk and linen fraying for the making of book-markers has been tried, but abandoned, for it required continual attention of the eyes in the selection of fine strands of material. Further, it is said that the practice fosters a tendency to destructiveness.

GROUP II—STANDARDS 3 TO 6; AGES 10 TO 13.

Following the order of work for the previous group marked *A* to *E*.

(A) *Bead Work* is practically stopped, for its educational value is small. Some few girls, however, carry on with it for the making of Japanese window screens. The fine bead work for making bags, &c., is necessarily barred.

(B) *Paper Work.*—This gives place to cardboard modelling, in association with lessons in arithmetic, language, &c. Similar articles to those made in paper are now made in cardboard.

(C) *Modelling in Clay and Plasticine* is continued for its expressional value in the teaching of history, geography, &c., and there is a beginning in the use of paper pulp for the production of permanent models.

(D) *Toy Making.*—Strip wood work and simple carpentry are begun and developed. The work is of value in the practice of making objects, and also in association with the teaching of arithmetic—e.g., the making of a barn according to the example of paper folding is developed, and the model barn is made of wood. Inches are taken as feet, and the barn measured: Base 20 by 14, height 12, roof 6 by 6. The wood required is measured up, and the cost found at 2d. a foot. Cost of paint at 1d. a foot. Cost of nails at 3d. a lb., allowing 2 oz. A great number of models are made such as ladder, fence, easel, swing, signal, notice-board, frames, drain, garden seat, deck-chair, and table.

(E) *Drawing*.—The systematic design drawing expands into bent iron work. This is an excellent form of handicraft for the short-sighted. For the most part it can be done with the eyes looking right forward at the work. With a bench or table of a proper height for the child there is no necessity for stooping, such as is involved in the use of carpentry tools. The work has drawbacks in that a good deal of noise is involved, and that in some of the advanced work the children have scarcely sufficient muscular strength to bend the iron unaided. The variety of this work done is as follows: Formation of C and S curves, joining by means of clamping, and by means of rivets, drilling, and fixing rivets. The elder children proceed to make such articles as watch stands, calendar stands, flower-pot holders, letter and key racks, photograph easels, flower glass holders, double photograph frames, oval frames, fire screens.

(F) *Utilitarian Crafts*.—In these higher standards work that was originally expressional becomes utilitarian, except in so far as the cost of the work can be worked out. Materials cost but little in comparison with the time expended, but this can be assessed and the practice of arithmetic associated with the more attractive handiwork. Knitting is continued. Lock knitting is applied to the making of hats, bonnets, muffs, necklets. Plain and purl in various patterns such as ribbing, imitation brioche, dice. Vests, scarves, tea cosies and jerseys are made. String bag making is developed. Casting on, passing stitches over, counting rounds, taking work off from frame, casting off, making cords for handle, putting on rings, and fastening off ends are taught and practised. The work does not involve eyestrain when the child shows a capability for it. They are not allowed to hold the work in their laps, it must be placed on the flat-topped myope desk. Raffia work is continued in association with cardboard work. It is useful as a mode of cultivating "taste." No raffia weaving or sewing is permitted, for these are far too trying to the eyes. Bookbinding of an elementary kind is begun. The children are taught to bind into folios the large printed sheets produced with the rubber-faced types in class work. Mat-making was tried, but stopped. It is a utilitarian work of the dullest and most routine kind, and has no immediate or prospective value for children of this degree of intelligence.

GROUP III—STANDARD 7; AGES 13 AND 14.

It is in this group that the greatest difficulty is found in providing a sufficiency of good handicraft such as can be done by highly myopic children. Carpentry would be ideal, were it not for the necessary stooping posture and the use of plan drawing and measuring. That prohibits the work for these children in anything like a full development.

Comparing their schedule with that of the former groups :—

(A) *Bead work* is stopped.

(B) *Paper folding* is developed on occupational lines: envelope folding, show-card making, and box making are taught. To some extent arithmetic lessons can be introduced by calculating the cost of material and time.

(C) *Modelling* is practically confined to the use of paper pulp; this has an educational value in the teaching of geography, and a utilitarian side, as the elder children may be able to produce permanent models for the use of the younger children, and for the blind schools.

(D) *Toy making* includes all the carpentry these children can undertake; many of them show considerable skill in the work.

(E) *Drawing* and design is developed in the bent iron work. It is proposed to attempt repoussé work and tinsmith work. Repoussé work, even more than bent iron work, has the disadvantage of the noise involved. Repoussé work as ordinarily done would not be permissible for the highly myopic, since the flat bench and the continual stoop over the work would be injurious. It would, however, be quite practicable for the work to be done on wall benches sloped at the angle of 30° from the perpendicular; the benches would have a 2 or 3-in. ledge on which to support the work and tools; the children would work standing before the wall benches, in very much the attitude of the sculptor. Tinsmith work would provide an additional exercise for the cleverer boys, but educationally it is no more than a development of the cardboard modelling.

(F) *Knitting* is continued by the girls. Bag making is stopped, but cane and basket work is continued. Raffia work is stopped, since the full development is prohibited for these children. Bookbinding is developed. The girls learn domestic work and simple cookery.

One of the best of the handicrafts for these older children is the printing, to which attention has been drawn. For value in the inculcation of thought, care, exactness, general neatness and cleanliness of

work, it is scarcely to be bettered. The materials employed are simple, and the work is variable to the ability of almost every grade of child. The only limitation is the one that confines the use of sticky black pigments to those who can keep themselves clean.

OCCUPATIONS FOR MYOPES.

In the former account of the myope classes some remark was made on the work of these children after school years. The necessary inquiries made by teachers and care committee members as to suitable occupations for those leaving school has resulted in the drawing up of lists of suitable occupations for boys and girls. It must be understood that this list relates to London conditions only. Two grades of work are shown, those most suitable and those of secondary suitability:—

Boys: First selection—Nursery gardening, poultry farming, messengers, assurance agents, travellers and canvassers, rent collectors, hawkers and street traders, shop work under good conditions, and piano tuning. Second selection—Stick and pipe mounting, basket making, some branches of brush making.

Girls: First selection—Florists' work, waitresses in tea shops, dairy shops, under nursemaids (no sewing), crèche attendants, helpers at mothers' schools, helpers at special schools and dining centres, and light warehouse work (packing). Second selection—Box making, cork sorting, envelope folding, cementing and black-bordering, show-card mounting, some branches of brush making, and stockroom work.

The kinds of work placed in the first and most suitable selection are of the outdoor type, or those that enable the worker to be out and about, standing and moving, and with a minimum of close eye work. These kinds of work are doubly suitable, for the general health is likely to be the better for the freedom of movement enjoyed, and with the improvement in the general health the state of the eyes is likely to be benefited. Next come the second selections. All these are light tasks, that do not unduly involve the use of the eyes, but they necessitate sitting and stooping to some extent and therefore are not so good for the body or the eyes. Looked at from the occupational point of view, it is evident that the teaching of handicraft in the myope classes is not a direct preparation for life work, such as is understood by the handicrafts of a blind school; rather the work should be regarded as a convenient peg on which to hang general instruction; the work itself is of value as a training in method and habits of precision, and cultivating

satisfactory habits in the use of the eyes. The work is of value also in that it tends to engage the minds of the children with the possibility of creating things and of fostering thought and observation, and to that extent withdrawing them from the fatal snare of living in a world of reading and book-thinking. The manual work is therefore both a discipline and a distraction.

If any comment be needed upon the satisfactory nature of these classes, it could best be found in the steadily increasing interest shown by the ophthalmic surgeons who have to do with the eyes of the elementary school children in the county in which the scheme has been most exploited. There is a steady increase in the number of children referred by the hospital surgeons "for education in a myope class."

DISCUSSION.

Dr. G. E. SHUTTLEWORTH said that, as a Fellow attached to the Sections of Psychiatry and Study of Disease in Children, he was grateful to the ophthalmologists for the opportunity of listening to the two papers by Mr. Leslie Paton and Mr. Bishop Harman. He was amused to find even here the usual divergence of opinion which he had invariably met with when he had attended meetings where the Montessori system was discussed. He was fortunate in having seen and conversed with both Dr. Seguin and Madame Montessori, and what struck him about each was their marked personality. With such personalities he did not think it mattered much what was the particular form of the didactic material used. He was pleased to hear Mr. Paton recognise, as Madame Montessori had done, that the apparatus was not altogether her original invention, but was based very much on what Seguin had described as long ago as 1846. When the Montessori furore was at its height he was present at a meeting when the apparatus was shown as a novelty, and he produced some sensation by stating he had used similar apparatus for thirty years, and Seguin had used it twenty years earlier than that. Mr. Bishop Harman seemed to consider that the perfection of all things had been reached in the special schools of London. Having had something to do with them for several years about 1900, he agreed that they were under most admirable management and organisation, but that was not owing so much to method and apparatus as to the personality of the lady in charge of them. A judicious infusion of the Montessori method into them would not, he thought, be to their disadvantage. Their methods had been largely derived from those of the Froebel system, but were happily tinged by the principles which Seguin laid down. He had himself given lectures to special school teachers on the principles laid down in the latter's treatises. With regard to the adaptation of the Montessori system for children with defective sight, he was unable to

speak from any close personal knowledge. He ventured, however, to think with Mr. Paton that some of the material shown was most excellent for training the tactile sense, and thus might be of service in educating the blind person's conceptions of dimension and in helping him to calculate. There could be no doubt that by teaching people (so to say) to see with their hands, marvellous results were obtained even when deafness and blindness co-existed, as was proved in the cases of Laura Bridgman and Helen Keller. He cordially joined in the thanks due to Mr. Paton and Mr. Harman for their interesting papers.

Mr. LESLIE PATON, in reply, said he had evidently rushed unawares into a controversial arena. So far as he understood Mr. Harman's paper, that gentleman had proceeded to describe, in fuller detail than he himself could have done, the development of a Montessori system for older children, an application of exactly similar principles to the special myopic classes. He would ask the meeting to remember that he specified his belief in the system for children in the pre-education period of their lives, before Council Schools received them; and for such he regarded the Montessori system as of very great value. He believed Madame Montessori was now striving to bring the system into use for older children. Until that scheme was developed and published criticism was out of place. When Mr. Harman said that the main education of children took place despite the Code (for it occurred through learning to appreciate by touch) he was supporting the contention which he (Mr. Paton) had been making, for Madame Montessori's system consisted in systematising this touching and handling. The method should be present in the minds of ophthalmic surgeons who had the chance of recognising defective sight in quite young children. It was well that ophthalmic surgeons should know of a system which enabled other senses largely to replace the defective sight. The learning of reading and writing was a mere incidental part of the system. It so happened that the faculty of writing was more readily and efficiently acquired than in other methods of training. The great point was the development of character and the power of concentration: teaching the child how to learn, and educating it in the widest and truest sense of the word.

Mr. BISHOP HARMAN, in reply, said he agreed with the speakers that the one important factor in education lay neither in the system nor in the technique of the system, but in the personality of the teacher. In the case of a young child with defective vision, even to blindness, the chief care of the parents should be to secure a good teacher who understood the nature of the defect and let that teacher look after the system.

Section of Ophthalmology.

Mr. PRIESTLEY SMITH, F.R.C.S., President.

December 2, 1914.

Case of Metastatic Suppurative Irido-choroiditis.

By H. ROBINSON, F.R.C.S.

PATIENT (F. P.), a married woman, aged 23, attended the Western Ophthalmic Hospital on November 3, 1914, complaining of inflammation and defective sight of right eye.

History: Four days ago had sudden attack of faintness and vomiting, with acute pain in head and right eye, lasting for two or three hours, after which she noticed that the eye was red and inflamed looking and the sight very misty. This got worse for two days, when she went to her doctor, who put in drops and told her to attend the hospital.

November 3: The right eye was generally congested, with much swelling and chemosis of the conjunctiva and slight muco-purulent discharge. There was little or no pain. Eyeball was tender to palpation. Slight exophthalmos. Ocular movements free in all directions. Pupil semi-dilated, fixed (? mydriatic). Cornea hazy and œdematous. Anterior chamber of normal depth. No hypopyon. Tension normal. Right vision, hand movements; left vision, $\frac{6}{8}$. Right fundus showed yellowish reflex; no details could be made out. Left fundus, normal. Urine: Specific gravity 1015, acid, no albumin, no sugar.

Patient was pregnant, five months, and had a considerable amount of vaginal discharge. Looked ill. Till the present attack had always had very good health and had no illness of any kind for many years.

The condition of the eye remained much the same for several days,

except that the congestion and chemosis gradually decreased. On November 16 the cornea was hazy and œdematous as before, but on November 17 it was perfectly clear, and it was then seen that the anterior chamber was extremely deep. The iris, deeply concave, shelved back towards a greyish-green opaque layer posteriorly, but leaving a distinct interval all round, in which the ciliary processes could be seen. On the concave surface of the iris, and near the posterior opaque layer, was a well-defined, complete ring of hæmorrhage. The position of the lens could not be made out. No P.L. Tension normal.

The case, which I take to be one of metastatic suppurative irido-choroiditis, presents one or two peculiar features—as, for instance, the complete absence of any sign of the lens, and the extraordinary rapidity with which the cornea in little more than twenty-four hours changed from being quite opaque and œdematous to its present condition of glass-like clearness and transparency.

June 9, 1915.

Case of Sympathetic Ophthalmitis treated by Intravenous Injections of Salvarsan and Neo-salvarsan..

By A. C. HUDSON, F.R.C.S.

F. B., AGED 19.

September 3, 1912: Left eye injured by a flying fragment while patient was chipping mosaic with a hammer and chisel.

September 14, 1912: Patient admitted to Royal London Ophthalmic Hospital, under Mr. Lang, suffering from left perforating corneal ulcer, with much infiltrated margins and adherent iris. Vision, P.L. Skiagram showed no intra-ocular foreign body. Right: slight injection; no K.P. Vision, $\frac{6}{5}$ partly.

December 16, 1912: Left irritable; pericorneal injection; T.—; no P.L. Right: sight misty six days; eye very irritable; ciliary injection; corneal haze, but no definite K.P.; posterior synechiæ; very dull fundus reflex. Vision, fingers at 2 metres. Differential blood count by Dr. Browning showed 9 per cent. of large lymphocytes amongst leucocytes.

December 20, 1912: Left, excision. Microscopic sections of a portion of the anterior part of the uveal tract of the excised eye showed *typical*

histological appearances of sympathetic ophthalmitis. Intravenous injection of 0.6 gm. "606" by Dr. Browning, at Mr. Lang's request.

December 29, 1912: Ocular injection much diminished; pupil widely dilated; vitreous opacity much diminished. Vision, $\frac{3}{60}$.

December 31, 1912: *Intravenous injection of 0.5 gm. "606."*

January 3, 1913: Vision, $\frac{6}{18}$.

January 11, 1913: Cornea hazy, but no definite K.P.; dull fundus reflex. Vision, hand movements.

January 14, 1913: *Intravenous injection of 0.6 gm. "606."*

January 17, 1913: Very little ocular injection; no K.P.; many posterior synechiæ. Vision, $\frac{6}{18}$ (one letter).

January 30, 1913 (case first came under care of exhibitor): Much injection; cornea hazy, but no definite K.P.; posterior synechiæ; dull fundus reflex owing to gross vitreous opacity. Vision, fingers at 1 metre. T.N.

February 4, 1913: *Intravenous injection of 0.9 gm. "914."*

February 9, 1913; Ocular injection much diminished. Vision, $\frac{6}{24}$ (two letters).

February 10, 1913: Ocular injection only slight; cornea bright; no K.P.; many posterior synechiæ, and lymph on lens capsule; moderate vitreous opacity. Vision, $\frac{6}{18}$.

February 27, 1913: Eye quiet; much lightly pigmented coarse K.P.; fundus indistinctly seen, no gross changes.

April 3, 1913: No fresh K.P.; media clear; fundus normal. Vision, $\frac{6}{6}$ partly.

April 24, 1913: Vision, $\frac{6}{6}$. Wassermann reaction negative.

Patient has been seen several times since the last note, the last visit being on April 4, 1915, when vision was $\frac{6}{6}$, and the fundus was normal.

Coloboma of Iris in Mother and Child.

By JOHN ATTLEE, M.D.

THE exhibitor said the interest of the cases lay in the colobomata taking an upward direction, whereas the common situation was downwards and inwards. In the mother it was completely upwards; in the child it was upwards and outwards in each eye, more markedly upwards in one eye than in the other. Webster Fox, a Philadelphia ophthalmologist, said in his book that coloboma upwards had been described.

Lieut.-Colonel ELLIOT said he had seen a good deal of coloboma of the iris ; congenital malformations were very common in India. He had never realized, when there, the importance of collecting statistics on this particular question. He had certainly seen coloboma upwards on several occasions, but had not taken the careful notes he might have taken as to its frequency.

Bony Growth in Frontal Sinus.

By CHARLES WRAY, F.R.C.S.

THE exhibitor said that bony tumours generally began in the frontal sinus ; and if a skiagram were taken of the specimen now shown it would be very apparent. The growth was the size of a bean. The peculiarity of the growth was that on one side it was hollow, and entered into the nose. If operation were contemplated, it would be considered to be a formidable osteoma. It did not present into the orbit. He also exhibited two other specimens, in one of which the section was made exactly under the cribriform plate. His reason for showing it was, that a number of ophthalmic surgeons operated upon bony orbital growths, and it was desirable that surgeons in the specialty should have absolutely clear notions of the anatomy of these cells. The other specimen showed the sphenoidal sinuses very beautifully. On the left side the sinus was very large ; on the right side it was smaller. The septum was deflected, so as to almost cover the sinus on the right side. Leading down from the bottom of the sphenoidal sinuses there were two very large cavities in the pterygoid process. Inflammatory conditions of the sphenoidal cells might cause retrobulbar neuritis ; and on looking at this specimen one could not help thinking that when cases were met with which were suspected to be of sphenoidal origin, and the symptoms were those of retrobulbar neuritis, it would be well to make the patient spend some time with the face downwards, so as to promote drainage from the sphenoidal and posterior ethmoidal cells.

DISCUSSION.

Mr. W. T. HOLMES SPICER said it was very difficult to make remarks about this specimen without further examination, but one feature which interested him particularly was that the exostosis, if it were such, had a cavity in it. He had had under his care for a number of years a case of an ivory exostosis

connected with the frontal sinus. He had sent the patient to Mr. Rawlings, at St. Bartholomew's Hospital, who removed it. But the patient now had a recurrence, and when her head was moved quickly to one side a splash of fluid was heard. Neither Mr. Rawlings nor he knew the meaning of it. If the formation of a cavity inside an exostosis were a probable occurrence the splash would be explained. The growth was now as large as a hen's egg.

Mr. CHARLES WRAY, in reply, said he would not call his case one of osteoma. He thought the explanation was that the patient had had a chronic inflammation of the sinus, and it had led to considerable thickening of bone, so that in some places it had reached a diameter of $\frac{1}{4}$ in. If a flap were simply turned down, and an examination made, one would infer that it was an osteoma. The condition was only discovered post mortem. He brought the case forward because specimens of bony tumours of the frontal sinus were extremely rare in pathological museums; there was only one genuine case even in the Museum of the Royal College of Surgeons.

Retinitis of Pregnancy.

By J. HERBERT FISHER, M.B., B.S., F.R.C.S.

YOU will observe that I have chosen for the title of these observations "Retinitis of Pregnancy," and not Albuminuric Retinitis of Pregnancy. I might, if I had desired fully to emphasize my own convictions, have headed it Toxæmic Retinitis of Pregnancy, but I want gently to lead you away from the idea, with which I fear ophthalmic surgeons are still satisfied, that the acute retinal changes in these cases are the result of the albuminuria and the kidney disorder. I presume we ally these cases with those of retinitis during acute nephritis after specific fevers or other causes. I confess that I do not myself see these cases of retinitis, and I imagine we should frequently be summoned by our colleagues to the medical wards if they were at all common and resulted, like the pregnancy cases, in rapid and very severe depreciation of sight. Our branch of the profession is perhaps getting behind the times in this matter.

Let me read you an extract from a paper published by Dr. H. G. Turney in the *St. Thomas's Hospital Reports*, vol. xxv, under the heading "Toxæmic Neuritis of Pregnancy." Bear in mind that this paper was written as long ago as 1896, and we shall, I think, realize that the ophthalmologist has some leeway to make up.

“It is largely to the work of Bouchard that we owe a clear appreciation of the fact that the life of the organism entails the constant manufacture of toxic products which, if that life is to continue, must be as constantly removed from the body. The main path of exit is by the urinary excretion, the degree of toxicity of which, therefore, should, other things being equal, form an index of the metabolic activity of the body. Thus it was found that the urine after severe muscular exertion was more toxic than that secreted after rest; that the urine of the day had a different toxicity from that of the night. If, however, the renal function was impaired, the toxicity of the urine was remarkably diminished.

“The corollary of this, the fact that the increased toxicity of the serum corresponds to a diminished toxicity of the urine, was furnished by Tarnier and Chambrelent and others. And now to the application of these facts. It was early observed by Naumann and Charpentier that the toxicity of the urine is largely increased in pregnancy, and that in certain cases the toxicity of both urine and serum may be augmented, this proving an absolutely increased formation of toxins. In eclamptic states the toxicity of the urine falls and the toxicity of the serum rises. As to the exact nature of these poisonous products which are constantly being evolved, we are still to a great extent in the dark. Bouchard proved that neither urea, uric acid, nor creatin was the active agent, though, as regards creatin, he has lately been contradicted by Duhrssen. The substances to which the toxic qualities should be attributed are, in Bouchard's opinion, the potassium salts and certain organic bases associated with the urinary pigment. These latter he failed further to isolate. Subsequent observers have largely added to our knowledge of the details regarding toxicity, but all that we can say with certainty is that more than one substance is concerned, and that it is a product of either anabolic or katabolic metabolism of proteid matter. It is this unknown substance (or substances) circulating in the blood that constitutes the toxæmia of pregnancy that is concerned in the production of uræmia or eclampsia, and that threatens us whenever from any cause either our metabolic processes get ahead of our excretory powers, or our excretory powers lag behind our metabolic processes.

“The frequent occurrence of albuminuria has been recently dealt with from this point of view by Professor Clifford Allbutt, and therefore requires but little comment from us. That, as he maintains, no theory of mechanical pressure either on the kidney or its vessels will account for this symptom there can be no doubt. To those who have accustomed themselves to regard it as the result of toxic influences, the idea that it is due simply to mechanical causes comes almost with the shock of surprise.

“Renal disease in the forms in which it occurs in connexion with pregnancy is *par excellence* a blood disease. In the early degenerative change described, for example, by Lindemann, as also in the later condition of cirrhosis, we see equally plainly the effects of toxins clamouring, so to speak, for excretion. It is noteworthy, too, that gross degenerative change may be present and yet the urine may remain free from albumin.

“Eclampsia may occur either with renal disease or without; it is impossible, therefore, to look upon it, as is so often done, simply as the result of renal disease; both alike must be regarded as common effects of the same poison. The significant condition of the serum and urine has already been described, and this renders further proof of the associated toxæmia unnecessary.

“Acute yellow atrophy of liver, a disease so intimately associated with the pathology of pregnancy, is now acknowledged to be the result of a very severe form of poisoning, probably introduced to the system by the portal circulation. Whether the poison is, as Hunter suggests, of bacterial origin and absorbed from the alimentary canal, we do not know, but we have to take that possibility into consideration before claiming acute yellow atrophy as an example of auto-intoxication. The pathogenic organism is still hypothetical, and till it is found we are justified in looking upon the morbid product as the result of some perversion of natural processes. The degeneration of liver cells which is found almost normally in the bodies of pregnant women, and which has hitherto been ascribed to anæmia, is almost certainly produced by toxic influences which are probably of the same nature as, but of less intensity than, those which are responsible for acute yellow atrophy. Bouchard has taught us that one of the most important, if not the most important hepatic function, is the arrest and neutralization of toxins arriving by the portal circulation.

“As with the kidneys, so with the liver; if the noxious bodies to be excreted arrive in overwhelming quantity, the excretory epithelium breaks down, and the process of elimination may be altogether arrested, with its usual disastrous effects upon the rest of the organism.”

The obstetricians have, I understand, modified their views in regard to many of the diseases of pregnancy and the puerperium and brought them into harmony with modern pathology. Albuminuria of pregnancy itself is no longer explained by them as the result of obscure reflex effects of the gravid uterus upon the kidneys, or by mechanical pressure on the ureters. Eclampsia they regard as totally distinct from the fits of ordinary uræmic convulsions—albuminuria of pregnancy, eclampsia, pathological vomiting of pregnancy, the obstetricians attribute to toxæmic products in the circulation. The outburst of eclampsia is a fulminating thunder-clap; it cannot be foreseen or predicted by any examination of the urine; no abnormal products in the urine which they can detect, no variation or deficiency in the normal contents of the urine will enable them to foretell it, though changes in the ratio between urea and the other nitrogenous elements may arouse anxiety. The toxæmia is something *sui generis*, its effects are found especially in the quality of the blood and in the blood-vessel walls. In fatal cases of eclampsia hæmorrhages in the brain are frequently, and hæmorrhages and necrotic changes in the liver almost constantly, found. That such

a vascular organ as the kidney, with its function of purifying the blood, should suffer in these cases is only what would be expected. Albuminuria is an effect not a cause of the trouble; in some cases a remarkable necrotic change of the whole kidney cortex, sharply delimited from the medullary pyramids, has been found. Such a toxæmia is surely the primary cause of the retinitis of pregnancy, a condition of the retina totally different in its onset, and usually in its appearances and certainly in its sequel, from what we know as albuminuric retinitis. But the obstetricians and pathologists have carried the matter further, though not yet to a final conclusion—they have endeavoured to find the nature of the toxin; though they have perhaps isolated nothing precise they have abandoned any idea of the poison being bacterial or the product of bacterial life. In the view which many of them accept, and which to me appears the most fascinating, they have found something and they have given it a name; they have called the poisons syncytiotoxins, by which they mean the products of the disintegration, presumably disordered disintegration—of the syncytium cells—such products have been found in distant parts of the body, and, speaking generally, I believe experimental pathological investigations have been made on these lines by the injection of cells and cell products into animals. But what are the syncytium cells? The two layers of cells, both of foetal origin, which form part of the developing placenta and lie next to the maternal mucosa, are Langhans's cells and the syncytium cells. Langhans's cells form an even layer of very ordinary cubical cells. The syncytium cells are so named from their immediately connecting the foetal and maternal structures; they rest upon Langhans's layer between it and the uterine mucosa; they are a layer of uneven plasmodial cells, all large but varying in shape and size, and many of them are multinucleated; they proliferate and form buds; these annectant cells cover the chorionic villi everywhere, but in the region where these villi enter into the formation of the placenta and invaginate the huge blood sinuses of this structure they are separated from the maternal blood-current, if at all, only by the single layer of thin endothelial cells, which line the blood-channels. Having performed their duty as annectant cells the syncytium cells in process of time disappear and no remains of them are demonstrable in the adult placenta. The products of their disintegration must be discharged into the maternal blood-stream, and if their disintegration proceeds on physiological lines no disturbance to mother or to foetus results. But it is known that the syncytium cells may be the subject of pathological processes—e.g., in

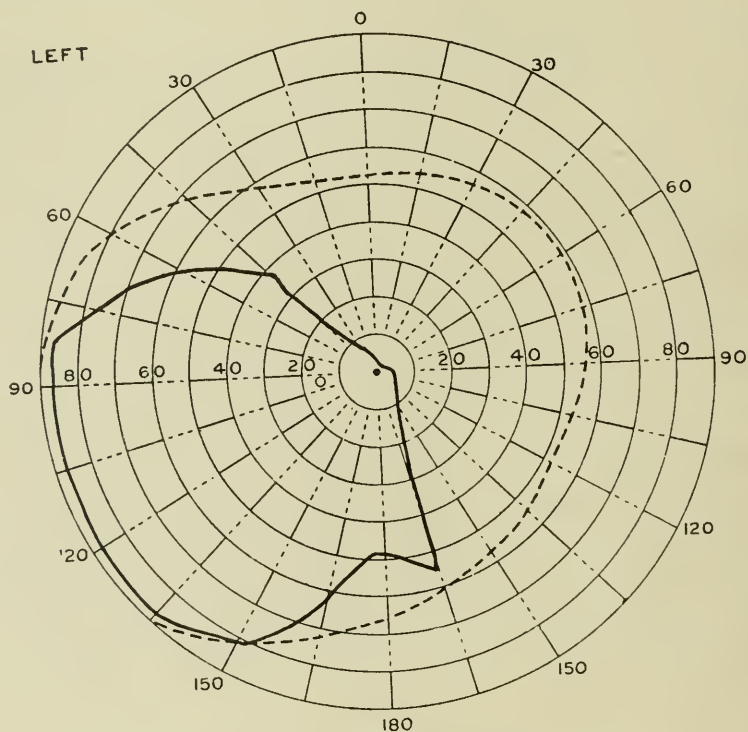
one direction they may give rise to the fatal condition of chorion-epithelioma; and being of fœtal origin, we here perceive an earlier example of matricide than is known to forensic medicine. If, as seems probable, owing to another perversion in their katabolism the syncytium cells give rise to the toxæmia of pregnancy which results in fatal eclampsia, an even worse charge lies against the fœtus, which commits the act of matricide by the insidious and deliberate method of poisoning. As this poison is not always present in sufficient dose to kill the mother, other severe disorders may arise, such as the pathological vomiting of pregnancy, non-fatal convulsions, albuminuria, and, in my judgment, toxæmic retinitis of pregnancy.

An elaboration of the above-mentioned views, first expressed by Veit, has been suggested. It has been put forward as an alternative that in the maternal blood there is produced an antigen for the destruction of the chorionic epithelial elements or even whole chorionic villi which are known to be shed into it. To this antigen to the syncytio-toxins the name of syncytio-lysin has been given, and it has been suggested that an excess of the syncytio-lysin and not the syncytio-toxins is the cause of the toxæmic diseases of pregnancy. This expansion of the idea does not carry us much further, but in support of it Leith Murray has found that the injection into rabbits and dogs of hæmolytic and agglutinative sera produces hepatic and renal changes similar to those found in eclampsia—it has been pointed out also that the same character of change in the kidneys and liver is found after death from snake-bite, and snake venom is known to contain both hæmolytic and hæmo-agglutinative toxins.

CASE I.

Mrs. R. T., aged 34, seen by me privately on February 4, 1908, was first known to suffer from albuminuria during pregnancy four years earlier, and this had since been repeated during pregnancy. The quantity of albumin was very considerable during her last pregnancy, which terminated at six months, four weeks before I saw her. The albumin had diminished in quantity, but was still present at the date of her visit. She was a lady who partook rather freely of alcohol. A month before the termination of her last pregnancy she observed that when looking at print it appeared to get muddled, and a week before miscarriage resulted, on closing the left eye, she found the sight of the right eye was defective—that she could not see the lower part of objects with it. She had always had good sight, and did not now specially complain of the vision of the left eye. The defect in the right eye was, however, still persistent. She was taking iron and was being kept on light diet. With the left eye she read

$\frac{5}{5}$ and J.1 well, but with the right $\frac{5}{12}$ only. The right eye in the ophthalmoscopic picture presented a large, well-formed, and complete star-shaped figure at the macula; there were many larger plaques of exudation with hæmorrhages on the front of some of them, and there were many smaller flame-shaped hæmorrhages, mostly in proximity to larger retinal vessels. In the left eye there were some large plaques and some finer dots of change, the latter being found in association with the lower temporal vessels and not far from the disk. There was no star figure, and no involvement of the yellow spot, in this eye. She was ordered to wear dark smoked glasses to protect her eyes, and was treated by her doctor on dietetic lines, due attention being given to the action of the skin, bowels, &c.



Mrs. R. T.—Field charted April 4, 1910. The loss is due to obliteration of retinal arteries.

She did not appear to obey the instructions thoroughly, but seven weeks after her visit I was informed that the pulse was less tense, that the albumin had disappeared from the urine, and that she had had two profuse periods.

I saw her again on May 19, 1908, when with the right eye she could read one or two letters of $\frac{6}{6}$, and with the left eye she read $\frac{6}{6}$. It was seen with the ophthalmoscope that all œdema had disappeared from the left fundus; there were no hæmorrhages; there were a few small gleaming spots running from below the macula towards the lower outer edge of the disk, and there were no arterial changes. In the right eye the O.D. was pale, the edges fluffy, the star figure was still present, and fine white spots were found extensively above its limits; here also only one hæmorrhage was seen; there was no œdema.

The next visit from this patient was two years later, on April 4, 1910, when I learned that she had been prematurely confined in January, 1909, but without disturbance of her sight. She again became pregnant, and was confined prematurely at six and a half months on January 8, 1910. In January, 1909, she had suffered from epistaxis, and in the early part of January, 1910, from hæmaturia. She had picked up a little since then. There was now (in April, 1910). albumin to the extent of one-sixteenth in urine. Her sight had failed about a fortnight before the last premature confinement. In the right eye the vision was now not any better than P.L.; with the left eye she saw $\frac{6}{20}$, or one letter of $\frac{6}{36}$, and picked out a word or two of J.8. There was conspicuous atrophy of each O.D., with pallor. The arteries of the retina, particularly in the right, were very small; the nasal vessels were the smallest, the superior temporal the best. In the left there was apparently equal atrophy of the O.D.; the vessels also were small, especially the temporal ones. There were gleaming spots in the macular region, but no hæmorrhage or active œdema.

I have been able to obtain the following account of the subsequent history of this patient from her doctor: The urine was never free from albumin, and the patient was always rather immoderate in her consumption of alcohol. She once more became pregnant, and was prematurely confined of a dead child at quite the end of 1910, or early in January, 1911. During this pregnancy the amount of albumin did not increase much. In June, 1911, her doctor was summoned to deal with very severe bleeding from the socket of an extracted tooth. After this she was left very weak and anæmic, and in August, 1911, went to a bracing seaside resort to recuperate. One day while sitting on the sea-front she suddenly gave a cry, and never spoke again, but rapidly passed into a state of coma and died of cerebral apoplexy.

CASE II.

Mrs. A. E., aged 39. Her sight began to fail at the middle of June, 1906, when she was six and a half months pregnant. She was suffering from albuminuria and was attacked with a fit in the night. The feet and ankles were swollen before and for two months after labour, which was induced by her doctor on June 30, 1906. She was more or less unconscious during the succeeding week and remembers no incidents in connexion with that time.

The doctor had been in attendance until December 4, 1906, and I saw her at St. Thomas's Hospital on February 18, 1907. Ever since delivery she had been liable to headaches and occasionally got lumbar pains. The feet and ankles continued to swell if she stood for an unduly long time. Her sight, she told me, improved after her confinement, and as far as she knew became as good as ever, but since Christmas, 1906, she thought it had been failing again. It seemed to her to vary a good deal. She had been measuring the quantity of urine passed and it amounted to only from 20 to 30 oz. in twenty-four hours. She had not suffered from vomiting but got attacks of giddiness, sufficiently severe to make it necessary to support herself or she

would fall. A fortnight before I saw her she had been liable to attacks, in which she became dizzy and inclined to lose herself. The vision in the right eye was $\frac{6}{9}$ partly and improved to $\frac{6}{8}$ with a $-0.75D.$ spherical. In the left the vision was $\frac{6}{1\frac{1}{2}}$ improved to $\frac{6}{8}$ badly with a weak sphero-cylinder.

When I examined her eyes in February, 1907, I found slight mottling in the macular regions, and one or two small and faint splashes of retinal hæmorrhage in each fundus. The disks were clear and there was no œdema of the retina. I found a little fine and one or two grosser spots of disturbance of retinal pigment in the peripheral part of each fundus. One or two of the small retinal arteries were of silver-wire appearance.

From a note which I received a few days later from her doctor, I found that she had suffered in her two confinements preceding that of June, 1906, from *post-partum* hæmorrhage, and was about as ill as she could be, and yet she recovered. The confinement in June, 1906, induced by her doctor, was complicated by puerperal eclampsia. At her worst she had about half albumin after the birth of the child. The quantity of albumin at the date of my examination of the eyes was about one-twelfth. Her medical man did not consider the heart either dilated or hypertrophied.

November 28, 1914: Her doctor writes: "I saw Mrs. E. to-day. She is in very fair health. She suffers occasionally from attacks of giddiness, never severe enough to cause her to fall. Every two or three months she comes to me for severe headaches, which yield to a purgative or two and some ammonium chloride. She had a bad attack of hæmatemesis about eighteen months ago. I diagnosed gastric ulcer, as she had suffered from dyspepsia for some weeks beforehand. She went into Lewisham Infirmary, but was not operated upon."

December 3, 1914: By arrangement with her doctor this patient attended in the out-patient department to see me to-day. I took her vision, and with a $+1D.$ spherical she read $\frac{6}{9}$ with either eye, and binocularly she read $\frac{6}{8}$, and with a $+2.75D.$ spherical J.1. Homatropine was put once into each eye to dilate the pupils for ophthalmoscopical examination, and within a few minutes the patient passed into a condition of coma and died in the casualty department to which she was removed. A specimen of urine was obtained and was found to be acid and to contain a large quantity of albumin, specific gravity 1006. No opportunity therefore was afforded me of determining the present condition of the fundus in either eye.

An autopsy made on the day following death revealed hæmorrhage of the ordinary hemiplegic type, which had occurred in the corpus striatum of the left hemisphere and burst its way into the internal capsule. The kidneys were found to be the subject of chronic interstitial change; they were of the ordinary red granular type and contained a few small cysts. The posterior half of one eyeball was secured for microscopical examination, and sections have been prepared and reported on by my colleague Mr. A. C. Hudson as follows:—

Pathological Examination by Mr. A. C. Hudson, F.R.C.S.

Mrs. E. Posterior half of eye fixed in 5 per cent. formalin.

Macroscopic Examination.—The coats of the eye are *in situ*. There is some œdema of the nerve-head. In the nasal quadrant of the retina are two large hæmorrhages. The retina at the macula lutea has a slightly puckered appearance.

Microscopic Examination.—Sclera and nerve-sheaths are normal. *Choroid*: The chief pathological changes are met with in the large choroidal arteries, many of which are the seat of gross changes of endarteritis which has in some instances produced obliteration of the lumen of the vessel. Nuclei are for the most part scanty in the proliferated tissue, which usually presents a homogeneous hyaline appearance. Not all the choroidal arteries are diseased, many of them presenting a perfectly normal appearance. Pathological changes similar to those seen in the choroidal arteries are met with in sections of posterior ciliary arteries surrounding the optic nerve behind the eyeball. There is a slight diffuse round-celled infiltration of the choroid. Bruchs's membrane is everywhere intact, and the retinal pigment epithelium *in situ* and of normal appearance. *Retina, nerve-head, and optic nerve*: Both retina and nerve-head exhibit evidence of œdema, which in the case of the retina is almost limited to the internuclear layer. In the nerve-head there has been considerable proliferation of supporting tissue, as is evidenced by well-marked hypernucleation. Nuclei of ganglion cells are present in normal number. Bodies of ganglion cells appear shrinking and rods and cones are almost totally absent, but it is not improbable that these appearances are due to post-mortem changes. The large retinal arteries exhibit changes due to endarteritis similar in appearance to those met with in the choroidal arteries; in the large retinal veins there is pronounced thickening of the adventitial coat. Changes of similar character to those met with in the retinal vessels are found, in transverse sections of the optic nerve, in the central artery and the collapsed central vein in the nerve-trunk.

CASE III.

Mrs. C. B. I saw privately on December 15, 1898; a married woman, aged 38, she was a mother of several children, of whom the youngest was aged 9. She had had no serious trouble in previous pregnancies. She had been operated upon for varicose veins five years previously, and it was known that she had no albuminuria at that date. She was pregnant at the time of her visit, the period of amenorrhœa being seven months, and her doctor confirmed her opinion. Four and a half months before I saw her there had been some uterine hæmorrhage; after this, she thought she began to pass more urine, having to get up at night for the purpose no fewer than three times. The quantity of urine passed was excessive, it was not a mere increase in frequency

of micturition. For one month she had noticed little clots of blood from the nose of a morning, but there had been no real bleeding. The sight first failed three weeks before her visit. Ten days earlier she could manage to read and was doing so for an hour at a time; her sight had failed rapidly. Her doctor noticed "albuminuric retinitis" about this date, and found the urine contained a large quantity of albumin; the specific gravity 1015, no visible casts; the quantity of urine was normal according to his report. I found no œdema of lids or lower extremities. The pulse did not appear to me to be tense. The vision in the right eye was less than J.20—the eye could not do better than count fingers at 4 ft. The left eye read $\frac{5}{12}$ indifferently, and words of J.4. The central portion of the right retina was œdematous; there were many spots of white exudation, mostly of circular shape, in this region, and also to the inner side of the O.D. All were of considerable size and there was a large plaque involving the fovea. One hæmorrhage only was seen, quite small in size, and situated up and out from the disk. There was no swelling of the disk and its margins were fairly clear. In the left eye the condition of the fundus was similar to that of the right, but the exudation spots were more abundant and more widely scattered, and there were no large plaques anywhere. There was no corona arrangement; there were several hæmorrhages in the retina in this eye. The appearances of the O.D. were similar to that of the right.

I heard from her doctor on December 23, 1898, that the patient was very ill with headache and vomiting on December 16 and 17, and that a quick and natural labour took place on the morning of December 20; the child was slightly decomposed, and had apparently been dead three or four days. The medical attendant had heard the foetal heart-beat on December 13, but could not hear it on either December 16 or 17. She was making satisfactory progress at the date of her doctor's letter. Though I saw this patient from time to time afterwards, I did not do so professionally, and never made a further ophthalmoscopic examination; but on May 24, 1899, her medical attendant, who had originally identified the "albuminuric retinitis," reported to me that she had made excellent progress, had very good sight, and that there was very little fundus change remaining when he examined the eyes about this date. There was still a trace of albumin in the urine, and he found the pulse a little tense, and slight hypertrophy of the heart. I happened to see Mrs. B. in October, 1899, and September, 1900, and in February, 1903, when she professed herself absolutely sound and well in every respect.

In November, 1903, her doctor informed me that she had been trying to do too much physically, and that there was some cardiac dilatation. Her eyes were said to be very good, but there was still a little albumin in the urine.

In December, 1903, she was recovering her usual health again, but she died on August 12, 1904, after being confined to bed for fourteen weeks "with a dilated heart, secondary to hypertrophy from chronic interstitial nephritis."

CASE IV.

M. B., aged 30, came to me at St. Thomas's Hospital on June 8, 1895, complaining of dimness of sight, "as if the eyes filled with water," the defect having been noticed six days in the right eye and three days in the left. The previous day she had complained of pains in the stomach, and had noticed puffiness of lower lids. She considered herself in the seventh month of pregnancy, and always suffered from swollen legs during pregnancy. There was slight œdema of lids. Vision: R., $\frac{2}{36}$ and J.18; L., $\frac{4}{60}$, with +1D. spherical $\frac{6}{60}$ and J.14.

Ophthalmoscopic examination: The disks were slightly swollen with œdema, to a height of 1.5D., and their outlines rendered indistinct. An œdema extended also in the retina; both above and below the yellow spot regions were numerous patches of whitish-grey exudate, of various shapes and sizes, in the retina, and some similar change was found in each retina to the upper and nasal side of the disks. A few scattered dots of hæmorrhage were found here and there, but they were neither numerous, nor large, nor typically flame-shaped. The arteries were tortuous and compressed and obliterated veins at points of crossing. There were no pigment changes, the peripheral part of each retina was fairly free from œdema, and there were no exudates in outlying parts.

The next day, examination of urine showed a large quantity of albumin; a specimen was faintly acid, specific gravity 1015.

I am sorry that I can say nothing as to the actual amount of albumin, or the total quantity of urine passed, and I have no record of an examination for casts. I learnt that she had suffered from epistaxis on four or five occasions during the last four months, but not from headache or vomiting.

On July 22 the pregnancy terminated spontaneously in a miscarriage, and the child was thought by the obstetric physician to have been of between five and six months' gestation.

I saw the patient again on September 7. She had no vomiting, headache, or epistaxis, no swelling of legs or eyelids, and except for the eyes and slight feeling of weakness had nothing to complain of. She appreciated no improvement in vision. There was, however, some gain, as with the right eye she saw $\frac{6}{60}$ and J.6; and with the left eye $\frac{6}{18}$, and even words of J.1 at very short range. There was now less œdema of the retina in the right eye, but still enough to obscure the outlines of the O.D. In the left eye the œdema of the retina was much less marked and the outlines of the O.D. almost clear. In this eye the retinal changes were sharply limited, ill-defined patches of exudation, mostly rounded, scattered over the central region of the fundus, and in the macular region more clearly outlined and brighter in colour; and in this part of the fundus there was some scattered irregular hæmorrhage. At the yellow spot there was an arrangement of glistening exudation in the shape of a half star or macular fan. In the right eye the dots of exudation were less numerous than in the left; there was the same half star of exudation at the

yellow spot as was found in the left, the points of the rays being directed towards the O.D. The urine at this time was acid, specific gravity 1010, albumin one-half. A week later the vision had risen in the right eye to $\frac{6}{18}$ partly, and J.1 at short range; and in the left to $\frac{6}{12}$ partly, and J.1 at 6 or 7 in. On September 11, 1895, the vision was about equal in the two eyes—namely, $\frac{6}{12}$ partly, and J.1 at fair range. The ophthalmoscopic note is to the effect that the condition now is like one of chronic renal retinitis. In each fundus at the yellow spot is a coronet-like arrangement of fine gleaming spots grouped in patches, and a few hæmorrhages. On the edge of these areas are a few more irregular and less shining spots of exudation. A few exudate spots were seen also to the inner side of the left O.D. In the right the outline of the O.D. is more blurred than in the left; in the latter it is sharper everywhere except at its upper and inner margin.

I last saw this patient on September 25, 1895, when the vision and ophthalmoscopic appearances were practically the same as a fortnight earlier.

CASE V.

Mrs. MacL., aged 32, was seen by me as a private patient on November 25, 1907. She was known to be the subject of albuminuria, and some previous pregnancies had been brought to an early termination on this account by her doctor. Years ago she had had some epileptiform attacks of uræmic origin. Eighteen months before I saw her she was confined in Japan; albuminuria was present, and she was "blind" for a considerable period. She came to this country in June, 1907, when her doctor found the urine solid with albumin. There were hæmorrhages in the retina, according to his observations, and there was dropsy of the legs. In September, 1907, the best vision was said to be $\frac{6}{100}$. Between each previous pregnancy the albumin had entirely disappeared. When I saw her in November, 1907, the quantity of albumin was about one-half. The vision of the right eye was less than $\frac{5}{60}$, and with this eye she could make out words of J.14. With the left eye she could read $\frac{5}{9}$ fairly, or even pick out a few letters of $\frac{5}{6}$, and on the near types could read J.4 or words of J.2. I found with the ophthalmoscope that each disk was obviously pale, the right rather more so than the left. The retinal arteries were conspicuously of the silver-wire type with degenerate coats and perivascular white lines in places near the disk. The inferior macular artery in the right eye was reduced to a white line only and was obviously obliterated. In each retina there were areas of retinal degeneration, widely diffused and extending to the periphery, and with pigment spots scattered over them. There were no hæmorrhages nor recent exudates. The field of the right eye to the hand test was not restricted.

She was not a patient who was very willing to follow her doctor's regime of treatment and diet, and especially rejected his advice to discontinue stimulants. I had the opportunity of examining her eyes again six months later.

There had been no improvement in vision and the ophthalmoscopic picture was in every way unaltered.

In this case I formed the opinion that the prognosis as regards both eyes and duration of life must be fairly bad. In reply to an inquiry I made, however, her medical attendant wrote me on June 5, 1915—i.e., seven years later—that “she is completely recovered and has been in excellent health during the past three years. Her eyes, too, are quite normal.” The statement in regard to the eyes no doubt implies that she has thoroughly serviceable sight in her left eye—the right is sure to be defective still, and if an ophthalmoscopic examination were made I anticipate that evidences of the past changes would still be found.

CASE VI.

Mrs. H. M. was seen by me under unfavourable conditions at her home in a suburb of London in June, 1900, one week after her confinement. The sight of the left eye went misty about six days before confinement and the right eye followed the same night, but was not as bad as the left. Its best vision at the time of my visit was only the ability to count fingers at 8 or 9 ft. Her baby was born dead at seven and a half months. She was a woman aged 35, and had had thirteen children, of whom eight were living. Twins had twice been born to her. No recovery of sight had taken place when I saw her and albumin was very abundant in the urine, but her doctor reported that it was getting less. The pupils were large; the right acted slightly better than the left to light. She was the subject of neuro-retinitis in each eye with hæmorrhage and large white exudates in the centre of each fundus. The heart was considered to be normal and there was no accentuation of the second sound. The pulse was rapid but not tense.

I saw the patient again on September 27, 1900, when the vision of the right eye was $\frac{6}{80}$, improved to $\frac{6}{36}$ with a -1D. spherical, and J.14; the left vision was $\frac{2}{80}$ and J.20. By this date the fundus changes were in a quiescent stage, but there were still many exudates, a few of them now showing pigmented change. The disks were pale, and the arteries reduced in calibre and their walls thickened. I saw the patient once again on November 19, 1900, when the vision of the right eye was $\frac{6}{36}$; with the left she could do no better than count fingers at 2 ft. The disks still showed signs of neuritis with blurred edges, and the right was filled in more than the left, but the left was the paler and its arteries were very small, less than those of the right. She considered herself now in good general health, but the urine was still not free from albumin.

CASE VII.

Mrs. E. C. was seen by me at St. Thomas's Hospital on December 7, 1910. She had been an in-patient in the Gynæcological Ward when labour was induced, mainly on account of the sight. She was suffering from retinitis and eclampsia. She was an in-patient for six and a half weeks. At the time I saw her she was still attending in the Out-patient Department under one of the obstetric physicians. The pregnancy, which was artificially terminated, was the sixth, not counting one miscarriage which occurred eight years previously. She had some swelling of the left leg during her first pregnancy, and both legs were swollen during the fifth pregnancy. There was only a trace of albumin in the urine at the date when I examined her eyes, and there had been no swelling of feet or legs since the end of September. She was pallid, but not feeble. She stated that the sight had greatly improved in the last three months. At first she could not see clearly at all with the left, but she could always read newspaper type with the right eye after her delivery. She still described the sight of the left eye as filmy, as if looking through water. Each eye had $\frac{6}{8}$ vision, with the pupils clear and acting well. After dilating the pupils I found some mottling near the macula in each eye, and a fine peppery pigment disseminated through each retina, with here and there a grosser clump of pigment change; there were no hæmorrhages, and there was no active retinitis. The retinal vessels showed no gross change, except in the case of the left inferior temporal artery; this artery was rather brilliant, and at its first division the descending branch crossed the corresponding vein and was somewhat clouded at the point of crossing, while the horizontal branch at once ceased to carry blood, and it was evidently occluded, being reduced to a white line.

CASE VIII.

L. H., aged 34, was seen at Moorfields Eye Hospital on March 31, 1908, the sight having failed in the previous seven weeks. The onset of the failure occurred three days before her second pregnancy terminated in the birth of twins. She suffered from swelling of legs, and the vision deteriorated to a faint perception of light. Nephritis was recognized by the local doctor, and when seen at Moorfields there were albuminuric stars at both maculæ, and a large exudate existed between the left disk and the yellow spot. There was some pigmentary disturbance in the lower periphery of each fundus. She had to be led about owing to defective vision for four months, after which time the sight gradually returned. In the following year, 1909, she gave birth to another child, but had no return of the eye symptoms. On August 23, 1913, the vision in the right eye with a + 3D. spherical was $\frac{6}{9}$, and in the left with similar correction $\frac{6}{12}$. The condition of the fundi at this date was as follows: There was no change in the yellow spot region in either eye. The disks were slightly pale, filled in, and were fluffy-looking at the margins. The retinal

arteries were small, but were not accompanied by white lines, and showed no signs of any sclerosis of their walls. At the periphery in both retinae, but more marked in the right, there were wedges and traces of pigment arranged roughly in a radial direction. The urine at this date had a specific gravity of 1003 and was acid, and there was doubtfully a faint trace of albumin discoverable by the boiling test; there was no sugar present. She was asked to report herself again in twelve months, but I have no record of her having complied with this request.

The case is of interest as showing the good recovery of sight after an unusually long interval of impairment as the result of retinitis of pregnancy, and the maintenance of sight over a period of at least five and a half years. It also afforded an opportunity of comparing the fundus conditions both during the stage of impaired vision and at a long date after recovery.

CASE IX.

Mrs. E. T., aged 28, came to me at Moorfields Eye Hospital on November 3, 1914. The sight had been misty for about ten days. She had four living children and now was seven months pregnant. The pupils were widely dilated with very slight action to light, and vision was not better than hand shadows. Ophthalmoscopic examination revealed a severe neuro-retinitis, with much exudation and large plaques in the central part of each retina, many hæmorrhages, mainly in the inner layers of the retina, and much general œdema. In the lower part of each fundus, the œdema was so extreme as to cause a detachment of retina, everywhere transparent, with a prominence as high as +10D.

I made arrangements for her removal to the Labour Ward of St. Thomas's Hospital, where, after a day or two in bed, labour was induced, and she was delivered of a dead macerated child on November 9. The amount of albumin at first in the urine was very large, sufficient almost to become solid on boiling.

I examined her eyes again on November 16, by which time the amount of urine seemed to be falling from above to something rather below the average quantity, and the amount of albumin which it contained was greatly diminished. My ophthalmoscopic note was that the right disk was decidedly clearer, the hæmorrhages were fewer, but there were still large plaques of exudation abundant in each. The left disk was less defined than the right, and there were more hæmorrhages than in the right retina. I thought the arteries slightly reduced in size. The œdema in the lower part of each fundus was decidedly less, but detachment was still made out in each, and was higher in the left than in the right, but even in the left it was appreciably less than a fortnight earlier. The vision was still not better than hand shadows and the pupils remained widely dilated. She thought she had slightly more sight in the right than in the left eye.

On November 30 the conditions in each retina had further subsided, and all appearances of detachment had now disappeared in each; the right disk was

still rather the clearer of the two. There were still exudates in the central part of each retina, but they were less defined than formerly; they appeared to merge more into a general diffused œdema. Hæmorrhages were still present in considerable numbers. By this time in the periphery speckled pigmentation could be seen, and in places larger clumps of pigment were visible; some of the pigment was found to lie in front of retinal vessels. There was still no material improvement in sight; the general health was satisfactory; there was still albumin to be detected in the urine but only as a very faint trace.

December 14, 1914: The vision is now improving, the pupils are smaller, and each is beginning to act better to light, but the direct sight is not better than the ability to count fingers at a foot or so with the right eye, while the left cannot yet count fingers. The Esbach test still reveals the presence of albumin in the urine, but it is less than half per cent., and scarcely detectable in the boiling test. There are still many hæmorrhages in each fundus. The arteries are undoubtedly reduced in size, and a branch of the superior temporal artery in the left retina above the macula is quite obliterated. All snowdrift exudates and all œdema have disappeared. Clumps of pigment, some of considerable size, are found in each retina over its whole area. The disks are by now fairly clear, though they have fluffy edges and pale surfaces.

By February, 1915, no further recovery of sight had taken place. Considerable atrophy of each optic nerve is apparent. All arteries are reduced in size in each retina, while the superior macular artery in the left and the inferior macular artery in the right eye have ceased to carry any blood. All acute changes have vanished; pigment clumps are widely scattered through each retina.

I find the above cases difficult to cast into groups. In some, observations after considerable intervals have been possible. Good recovery of vision is the rule, accompanied by disappearance of all acute manifestations in the retina, and in this respect they differ absolutely from ordinary renal retinitis cases; some peripherally situated pigment disorder is usual and not surprising in view of the intense and widely extending general œdema of the retina in the acute stage. Where defects of vision are permanent it appears to me, judging from my own cases, to depend chiefly upon changes in the walls of the retinal arteries, or on an atrophic condition of the nerve which, if it be an ascending atrophy, may well be secondary to changes in the ganglion cells resulting from imperfect blood supply. We have some reason to believe that the toxins of pregnancy, whether they be syncytio-toxins or syncytio-lysins, exert their effects directly upon the blood and the blood-vessels.

I am sorry that I have not been able to trace out the after-history of a greater number of my cases. They may live at least for many years—a recurrence of retinitis is by no means necessary, perhaps not even

probable, if subsequent pregnancies take place. In some the albumin appears to disappear entirely, but not in all cases; this does not to my mind prove a difference of causation or a necessity for differential classification. The kidneys are very vulnerable to the toxins of pregnancy; in some cases the damage done to kidney structures may be slight, transient, and recoverable; in others it may be so severe that only temporary amelioration is possible, with slow progressive changes to follow. In some of the worst cases which terminate fatally in the acute stage it has been proved at autopsy that the renal cortex is converted into little better than a slough. One of my cases (Mrs. C. B.) died after an interval of nearly six years from cardiac failure; another (Mrs. A. E.) died recently after eight and a half years, in tragical circumstances, of cerebral hæmorrhage into the left corpus striatum and internal capsule. The posterior portion of one of the eyes in this case was obtained and pathologically examined, but unfortunately I was deprived of the opportunity, which I so nearly realized, of an ophthalmoscopic examination immediately before her death. The kidneys were found to be of the typical red granular type with a few small cysts—chronic interstitial nephritis of the ordinary type. A third case (Mrs. R. T.) died suddenly from a cerebral apoplexy. In this instance albuminuria associated with pregnancy was first recognized seven and a half years before death; it recurred with subsequent pregnancies, and probably in this case albuminuria *may* have persisted in the intervals between pregnancy. Her vision was first affected in a pregnancy three and a half years before her death, and retinitis was seen by me in the active stage one month after this pregnancy had terminated in premature confinement. Full recovery of vision took place, and albumin was said to be absent from the urine two months after I saw her. She again became pregnant and was prematurely confined a year later, but without disturbance of sight; at the end of this pregnancy she had severe epistaxis. Eighteen months before her death a premature confinement at six and a half months was accompanied by hæmaturia; vision had failed fourteen days before this confinement. Albumin was present when I saw her three months later; there was then no active retinitis, but considerable atrophy of optic nerves, and striking changes in the retinal vessels. Albumin certainly did not disappear after this date, and one more pregnancy ended prematurely: during this pregnancy the amount of albumin did not increase much. In this case the renal changes were present and were probably progressive for seven and a half years, yet the retina was attacked during only two of her many

pregnancies. It seems to me that it is essential to the explanation of such a case to introduce the conception of another exciting cause for the retinal changes. I suggest that such a cause is the toxæmia of pregnancy. I do not presume to offer an opinion as to whether the toxins of pregnancy are capable of producing alteration in the retina independent of the presence of the renal changes, but the case goes some way to prove that albuminuria of pregnancy is not in itself sufficient to set up the typical retinitis of pregnancy. Another patient known to have had albuminuria in pregnancy probably something like twelve years ago for the first time, necessitating the artificial termination of pregnancy for epileptiform attacks, is still alive and in excellent health according to her doctor's statement, the sight also being well preserved in one eye.

DISCUSSION.

Mr. J. STROUD HOSFORD said he brought two cases of the condition because they provided him with an opportunity of commenting on what he regarded as the feature of fatality of the retinitis of pregnancy if the history of the cases were followed for ten to twelve years. During fifteen years he had carefully observed sixteen cases, of whom three only were now alive. Of the thirteen who died, two had a little sugar in their urine and hæmorrhages into the vitreous. All the cases except two ultimately developed a star-shaped spot at the macula, and that he regarded as a very important point in diagnosis and as an indication of a likely fatal termination. The history of one of his cases showed how the patient might apparently recover and remain well for a time. Her age was 24, and her first confinement was on November 6, 1900. There was albuminuria during the last four hours of her pregnancy, as her doctor reported, and in the first stage of labour she had a convulsion. On coming out of that she was blind for twenty-three hours. He saw the patient fourteen hours after confinement, but found nothing wrong in either fundus. His diagnosis was the amaurosis of pregnancy. She made a good recovery, and had two children afterwards, no trouble occurring with the urine according to her doctor's report. But in 1907 she attended at the Royal Eye Hospital with atrophic albuminuric retinitis, with stellate spots at each macula. Vision was only $\frac{6}{80}$ in the right, and perception of hand shadows in the left. She died suddenly in 1909. So it was seven years before she had real evidences of kidney trouble. One of his other cases had paralysis of the sixth nerve, and she also died of nephritis six years later. In another case, after a long time, the eye became proptosed, and in one eye there was atrophy of the optic disk: her death was from cerebral apoplexy. Mr. Hosford was of the opinion that this followed hæmorrhage into the sheath of the optic nerve as was recorded by Duke Charles Theodore, of Bavaria, who published an elaborate examination

of one of his cases in 1887. Of 132 cases of ordinary Bright's disease, most of which were seen in Dr. Fenwick's wards, eighty-one were in females, fifty-one in males. He thought the Section might very well make an investigation into the reason of the greater prevalence of renal retinitis in women compared with men. In the cases he had observed, it seemed that among single men and single women the rate was nearly equal; the female excess being caused by cases among married women, so the amount of renal trouble seen afterwards might have started in pregnancy. He had tested the refraction of the 132 cases mentioned, and in only two were the eyes myopic: there was nothing beyond slight hæmorrhages in them, not the least typical of renal nephritis, and those patients were still alive. The single case in a myopic eye in which he had seen anything like real albuminuric retinitis, or any definite retinitis, was in a private gentleman who had -4 and cylinder 2, who died of uræmia ten months after eye symptoms appeared. He therefore concluded that the condition of retinitis was very rare in myopic eyes, though he could offer no explanation of that. Since the introduction of the Wassermann reaction, he had had all the recent cases tested, but none of them gave a positive reaction.

Mr. W. C. ROCKLIFFE said he hardly gathered from the paper the treatment advocated by the author. He had only seen a few cases of retinitis in pregnancy. In one of these a woman in her first pregnancy developed symptoms of retinitis albuminurica in both eyes during her sixth month, the condition apparently proceeding to atrophy of the nerve. Labour was prematurely induced, and she recovered her sight to $\frac{6}{6}$, and no sign of fundus changes remained. Two years later she had a second pregnancy, and had almost a repetition of the eye condition. On his suggestion a premature termination of the labour was carried out, but her sight this time did not recover, and the condition progressed to blindness from complete atrophy and remained so. She died four years later from heart failure. Another case was that of a woman, whose first child was born in 1898, when there was nothing unusual. A second child was born in 1903, and she began with symptoms of ordinary albuminuric retinitis, which proceeded to blindness in one eye. Premature labour was induced, and she recovered her sight and when he saw her again, a year later, the fundus was normal. She died five years afterwards, it was supposed, from heart failure. Hitherto, he had presumed, with the old school, that the condition was due to mechanical pressure on the ureter, and that when this was relieved the patient usually recovered.

Dr. ATTLEE asked whether it was a fact that these cases were commoner among the poorer classes; and, if so, was there any explanation advanced for it?

Mr. J. B. LAWFORD said he thought one of the points of some importance which had been brought out in Mr. Fisher's paper, and which had also come under his own observation, was, that whatever might be the nature of the toxæmia its effects were very far-reaching. Mr. Fisher mentioned that in

several of his cases, in spite of the fact that during subsequent pregnancies there was no recrudescence of the retinal change, toxic effects showed themselves at a later date, and ultimately led to a fatal result. He was somewhat surprised to hear the author say that repeated pregnancies did not appear to exert much influence on the retinal conditions. He thought it had been a common belief that if a woman had retinitis during pregnancy, one of the safeguards against a recurrence was to avoid a subsequent pregnancy. It seemed, however, that measure of precaution did not, in itself, prevent the ultimate effects of this toxæmia. A case in point, which came under his own observation some years ago, was that of a lady aged about 25 who, during her first pregnancy, developed well-marked retinitis, with a great defect of sight. The symptoms and signs all disappeared after delivery, which he believed was induced prematurely—he was not sure, as the case occurred more than twenty years ago. She was regarded as cured. The doctor in charge of the case held the view—with which he (Mr. Lawford) agreed—that it was extremely inadvisable for her again to become pregnant, and she did not; and she went on with good sight for many years. But ultimately her sight failed, with atrophic changes in the optic nerves and retina, and she ultimately died of chronic nephritis. Another case under his observation was that of a woman somewhat older, who developed retinitis during her third or fourth pregnancy, with some reduction of vision, without eclampsia or severe general symptoms. She was allowed to go to full term in her pregnancy, and delivery of the child was followed by recovery of the ocular lesions. The same advice was given as in the former case, that there should not be another pregnancy, but it was not followed. Although at the subsequent pregnancy the retinal changes were apparently not at all increased, the general toxæmic condition was very much aggravated indeed, and the patient died shortly afterwards of chronic nephritis. He would like to know what the experience of obstetric physicians was with regard to the persistence of the toxæmic effects in these cases, and whether there was any adequate explanation of the persistence of these effects, even in the absence of subsequent pregnancies. He thought it was necessary to alter one's views as to what used to be called the comparative harmlessness of the retinitis of pregnancy.

Mr. LESLIE PATON considered that the paper just contributed by Mr. Fisher was one of great importance to ophthalmic surgeons. In these times, he thought, when many people were inclined to insist too much on bacterial causes for disease, this paper brought one to a better sense of proportion in the study of disease generally. For here was a disease of very severe character in which it seemed certain, as Mr. Fisher had said, that the toxin was not due to a germ at all, but originated in the syncytial cells. The disease under discussion was presumably caused by a cyto-toxin; and he thought certain other diseases would ultimately be brought into the same category. He would suggest, very tentatively, that certain types of irido-cyclitis seen occasionally in women at the climacteric might be due

to devolution taking place in tissues at that stage of life. If the paper brought about a realization of the fact that degenerative cyto-toxins might set up inflammations, it would have done great service. Another point of great interest about the cases recorded by Mr. Fisher was that the serious results exhibited in them were largely vascular—i.e., the seriousness of the disease seemed to depend on the extent to which the vessels were injured at the time the toxæmia was manifested. One's judgment as to the severity of a case must largely depend on an examination of the vessels. He was thinking particularly of the case of one patient he saw who had a very severe toxæmic retinitis in pregnancy, and in whom the vessels were not seriously injured; some small silver-wire-like vessels could be seen. It was now eight years since that illness, and the patient still remained healthy; all the residue she had was slight cardiac hypertrophy and slight silver-wire arteries. If the vessels were not seriously injured at the date of the first attack, the prognosis of the case was not very bad. But he was specially interested in the statement of the author that subsequent pregnancies need not necessarily cause a recurrence of the retinitis. One was occasionally asked an opinion on that question. Within the last fortnight he had been asked whether a subsequent pregnancy was permissible in such a case if health was to be preserved. His reply was that he did not think there ought to be one; but, though the family doctor acquiesced, he feared that the desire for another child would over-rule his recommendation. He therefore felt somewhat comforted by Mr. Fisher's statement.

Mr. HERBERT FISHER, in reply, said he was gratified by the interest his paper had aroused. Mr. Hosford, in his remarks, suggested that there was a greater proportion of cases of renal retinitis discoverable among married women than among either single women or male subjects. If so, that pointed to something peculiar to the married state in the female, and supported the toxæmia idea, as opposed to what had formerly been dwelt upon, the supposed albuminuric cause for the retinal changes of pregnancy. Mr. Hosford's case, in which there was paralysis of the sixth nerve, indicative of peripheral neuritis associated with pregnancy, a condition well known in neurological medicine, also supported the idea. He had himself many years ago reported some cases of ophthalmoplegia interna associated with toxæmia of pregnancy in the *Ophthalmic Review*. In answer to Dr. Attlee's question, speaking roughly from his own figures, he saw no justification for the belief that the condition under discussion was more frequent among the poorer classes; he thought the reverse was more likely to be true. In reference to treatment, a point on which Mr. Rockliffe asked information, many of the cases he had seen had been taken into hospital with the view of inducing premature labour; but most of the patients had aborted without professional assistance. It was certain that a viable child was not likely to be produced by a patient suffering from such severe toxæmia as to cause gross changes in the retina, and it was sound advice to terminate the pregnancy. With regard to what advice should

be given when one was asked whether further pregnancies should be allowed, when the patient was known to suffer from the retinitis of pregnancy, he would point out that in several of his cases there had been a series of pregnancies; in some deterioration of sight and gross changes in the retina had taken place, but intervening pregnancies had frequently been attended by no further damage to the eyes. So there seemed no certainty that a subsequent pregnancy would, of itself, be dangerous to the eyes. If the pathological view he had stated was correct, that the changes were dependent on perverted catabolic processes in the foetal structures, this would be a question in which obstetric physicians could give help; these changes could set up a variety of troubles in brain, kidneys, liver, and eyes. If the processes of subsequent pregnancies were carried out in a normal physiological manner no additional toxins would be poured into the maternal circulation, and there seemed no reason to anticipate further damage to the patient's eyes. To arrive at a conclusion it would be necessary, with the aid of obstetricians, to consider all the diseases attributable to the toxæmia of pregnancy, and not deal with the retina alone. He agreed with Mr. Paton's remarks, and indicated the fact in the paper that the vascular changes were of very great importance. The general evidences were in favour of the toxins of pregnancy attacking the blood or the vessel walls, and this was strikingly supported by the hæmorrhagic condition of the liver in pathological pregnancy and by the cerebral hæmorrhages often found in patients who had died of the toxæmia of pregnancy. And in estimating the chances of recovery in a given case, stress should be laid on the condition and size of the retinal vessels. In many instances those vessels were obliterated; in others they were reduced in size. Where there was much vascular constriction there was permanent loss of vision; but if the calibre of the retinal vessels was well preserved a good recovery might ensue, for if the general nutrition were kept up the exudation and œdema might be expected to subside.

APPENDIX.

The Definition of Blindness.

BEING A REPORT PREPARED AT THE REQUEST OF THE LOCAL GOVERNMENT BOARD DEPARTMENTAL COMMITTEE ON THE WELFARE OF THE BLIND — BY A SPECIAL COMMITTEE APPOINTED BY THE COUNCIL. AMENDED AND ADOPTED BY THE COUNCIL, 21ST JULY, 1915.

BLINDNESS is, strictly speaking, the inability to distinguish light from darkness. This definition is precise, but too exclusive for the purpose in hand, which relates to the Education, Employment, and Maintenance of the Blind. Many persons who can perceive light, and in some degree the form of objects, are yet practically blind as regards the ordinary activities of life, and it would be unreasonable to withhold from them such aid as is given to the totally blind. The task of the Committee, therefore, has been to consider what classes of persons should be regarded as *practically blind*, and how they may best be distinguished from the rest of the community.

In the first place, it must be pointed out that it is impossible to frame any precise definition of practical blindness which would suitably apply in all cases. It is necessary at the outset to adopt different standards for children and for adults, for the special needs of the afflicted children relate to elementary education, while those of the adults relate to industrial or professional training or to the granting of monetary aid. These must be separately considered.

As regards *children* an authoritative rule is already in existence. It relates to elementary education. The Act of Parliament entitled "An Act to make better provision for the Elementary Education of Blind and Deaf Children in England and Wales" (56 and 57 Vict., Ch. 42, September 12, 1893, Section 15), says: "In this Act the expression 'blind' means too blind to be able to read the ordinary school books used by children."

This rule appears to work satisfactorily. It is obviously wanting in precision, but this is not necessarily a fault. It would be possible to supplement it by laying down numerical standards, but if this were done it would be necessary to insist that these standards should not be rigidly applied. The reason is that there are many children, e.g., those

with high myopia, whose visual acuity is fairly good, but who yet cannot follow the ordinary school course without further loss of sight.

The Committee, therefore, does not recommend any modification of the rule already in force under Act of Parliament with regard to children. It desires, however, in this connexion to draw attention to three matters of practical importance:—

(a) For the safe and suitable education of the afflicted children it is not sufficient to class them according to *degrees* of blindness only; it is necessary to distinguish the *nature* of the defects. This can be done only by a person who in addition to medical training has had special experience of disorders of the eye. It is therefore important that the authorities should, whenever possible, entrust the duty of classifying the children in this respect only to persons known to be so qualified.

(b) For children who are too “blind” to read the ordinary school books, but yet too “sighted” for a Blind School, where eyesight is hardly used at all, and where reading is only taught by the Braille method, there is at present in many places no special provision. Where there is no sufficient need for a separate so-called Myope School such children should be taught in Special Classes in the ordinary schools. Local authorities have power to make this provision. More attention might well be given to the matter.

(c) According to the Act of Parliament the children whose needs are best met by the Special Class or the Myope School are classed as “blind.” But they are not blind in the ordinary sense of the word. In dealing with them the term “blind” should be avoided as far as possible. It places a stigma on them which their condition does not justify, and raises opposition on the part of parents to their education by the methods which are best for them.

As regards *adults* there is at present no authoritative rule as to what persons should be regarded as practically blind. The standards adopted by philanthropic agencies are far from uniform. As used in the Census the term “blind” carries no precise meaning. This is not surprising, for the only precise definition of blindness which can be given is that which stands at the beginning of this Report, whereas for the practical purposes of Social Economy and Philanthropy a wider meaning must be given to the term.

In the Bill now before Parliament to provide for the Technical Education, Employment, and Maintenance of the Blind, the following

definition is given (p. 6): "In this Act the expression 'blind' means too blind in the opinion of the Local Authority to perform work for which eyesight is ordinarily required."

This definition follows the principle already in operation under the Act relating to blind children. It states no precise standard, but leaves the responsible authority free to judge each case on its merits. The Committee is of opinion that this principle is the right one, and that the definition given in the Bill should be adopted, subject to the substitution of the word "essential" for the words "ordinarily required." The need for this substitution may be shown by an example:—

A blind typist or pianoforte tuner performs "*work for which eyesight is ordinarily required.*" Therefore, according to the definition in the Bill the term "blind" does not apply to him. Let the definition read "*too blind to perform work for which eyesight is essential,*" and his case is covered.

The Committee has carefully considered the advisability of supplementing the foregoing definition by numerical standards expressing degrees of blindness. Experience shows that persons whose acuity of vision (refractive error being corrected) is below *one-twentieth* of the normal ($\frac{3}{60}$ Snellen) are usually unable to perform work requiring eyesight, while persons with vision better than *one-tenth* ($\frac{6}{60}$ Snellen) are usually able to perform some such work. Persons with intermediate degrees may or may not be able; much depends on intelligence and bodily strength, and much on the nature of the blindness. A person whose so-called blindness depends on defects in the centre of the visual field may fail to reach a given standard and yet be able to perform some kinds of work requiring eyesight, while another person suffering from great contraction of the field of vision may surpass the same standard and yet be unable to walk alone or to do any kind of work requiring eyesight.

The Committee is of opinion that the numerical limitations mentioned above are likely to be useful as preliminary guides, but that until experience has been gained through the working of the Act they should be regarded as purely tentative. The certifying authority should not be bound by any precise numerical standard.

By what method are applicants for benefit under the Act to be examined and certified as eligible?

The Bill provides that the word "blind" shall mean "*too blind in the opinion of the Local Authority,*" &c., and it defines "Local Authority" as "the Council of any County or County Borough." It

does not prescribe the method by which the Local Authority shall arrive at an opinion. The Committee regards this question as one of great importance in relation to the equitable working of the Act. It suggests that the method should be laid down by law and that in principle it should be as follows:—

(1) Every applicant for benefit under the Act should be duly examined and certified as eligible or ineligible according to the provisions of the Act, by a person or persons approved for the purpose by the Local Authority.

(2) The Local Authority should approve for this purpose persons only who are registered under the Medical Act and who produce evidence of possessing competent knowledge of diseases of the eye.

(3) In every case a certificate should be given on a form provided for the purpose. The certificate should state in detail the facts elicited by the examination (including, if possible, the original cause of the blindness), together with the grounds on which the applicant is certified as *eligible* or *ineligible* for benefit under the Act. It should also state whether the case should or should not be *re-examined* at a future time.

(4) All such certificates should be subject to supervision by an Inspector or Assessor appointed by the Central Authority or Secretary of State. There should be a right of appeal to the Inspector in any case in which the justice of a certificate is called in question, whether in the interest of the applicant or of the public funds.

The foregoing suggestions are based to some extent on the regulations already in force under the Acts relating to Mental Defects. Certification of the mentally affected is carefully safeguarded in the interest of individual liberty, in order that no person may be brought under the provisions of the Acts without good reason. Certification of the blind will need to be safeguarded not only in order that the genuinely afflicted may receive their due, but that improper claims may be rejected.

Experience shows that many persons are willing to exaggerate their visual incapacity, and some even to feign blindness where none exists, in the hope of obtaining monetary or other advantage. Insurance Societies habitually submit claims of this kind to the judgment of an ophthalmic surgeon before agreeing to pay compensation. Exaggerated and dishonest claims are unfortunately not infrequent. In some cases the attempt to deceive is detected easily; in others, only by means of elaborate tests. Every member of the Committee, and

probably every ophthalmic surgeon, could cite cases of the kind from his own experience. Here are three examples:—

A young woman professing loss of sight through accident was granted compensation at the rate of 4s. 6d. a week. She received it for seven years. She received in addition much charitable aid. She was for three years an inmate of a blind institution. Ultimately the Insurance Company, being suspicious, demanded a trial. By order of the judge the patient was placed under the observation of an ophthalmic surgeon and a physician. She was proved to be a malingerer. A severe reprimand was administered by the judge and the compensation was withdrawn.

A young man with imperfect sight obtained a pass permitting him to travel by tram-car free of charge. It was given to him by a non-medical official of a Society for the Blind. He was found to be in the habit of riding a bicycle to the place where he joined the tram-car.

A man in jail, awaiting trial for burglary with violence, awoke one morning—so he asserted—completely blind. He appeared unable to dress or feed himself. He groped with his hands when made to walk alone. The prison surgeon suspected malingering but could not prove it. An ophthalmic surgeon demonstrated it by means of the prism test. (When both eyes are in use a suitable prism held in a suitable position before either eye leads to an involuntary movement of the eye for the avoidance of double vision.) The man was told that his blindness would disappear as suddenly as it came, and that its continuance would be likely to get him into further trouble. It was gone within a day or two. This man was a dangerous criminal and had been several times convicted. No doubt he hoped that blindness would excite compassion at the coming trial and mitigate the sentence.

It is hardly necessary to point out that the benefits which it is now proposed to confer upon the blind will offer to many persons a new and great temptation to exaggerate their incapacity, and that the establishment by law of an efficient and impartial method of examining and certifying applicants is a matter of far-reaching importance.

The Committee has suggested that the certificate should state, whenever possible, the original cause of the blindness. The chief reason for such statement is that the statistics obtained in this way would prove valuable in relation to measures for preventing blindness.

It has also suggested that the certificate should state whether re-examination at a future time is, or is not, desirable. In the large majority of cases probably a single thorough examination will be sufficient and final, but in some a subsequent examination may show recovery of sight—e.g., cases in which a corneal opacity clears spontaneously with lapse of time, and cases of cataract in which good

vision is restored by means of operation. On the other hand, persons who are certified as ineligible when first examined may be found eligible at a later time.

A question of importance remains to be considered. Should assistance be given under the Act to persons who wilfully and unreasonably refuse or neglect treatment which is likely to restore their sight? Should it be given to those who wilfully perpetuate or even cause their blindness by their own act? Such persons are not numerous at present, but they are likely to increase in number unless they are excluded from the benefits of the Act. Neglect of timely treatment in cases of eye disease is already far too frequent. If such neglect should carry with it a reward of 10s. a week for life it is likely to become more frequent. Again, among those who become incapacitated through "tobacco blindness" there are already a few who deliberately choose to remain idle rather than to give up their tobacco. If monetary relief be given in such cases their number will certainly increase.

In the opinion of the Committee the benefits of the Act should not be extended to persons who unreasonably refuse to take measures for the recovery of their eyesight.

In urging that the administration of the Act should be carefully safeguarded the Committee has in mind not only the waste of public money which would otherwise occur, but also the harm which would be done to the character of the persons concerned. In order to do the maximum of good and the minimum of harm the persistent aim of those who administer the Act should be to provide useful occupation for the afflicted persons and to help them, whenever possible, to help themselves.

PRIESTLEY SMITH,

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Royal Society of Medicine.*

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*Honorary Secretary of the Ophthalmological Section
of the Royal Society of Medicine.*

I

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE EIGHTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1914-15

OTOLOGICAL SECTION



LONDON
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1915

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OTOLOGICAL SECTION.

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Otological Section.

November 20, 1914.

Dr. ALBERT A. GRAY, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

GENTLEMEN.—Before we begin the more important business of the meeting I may, perhaps, be allowed to make a few introductory remarks.

I wish, first of all, to thank you for the honour you have done me in appointing me President of this Section.

Our meetings this year have come at a time of intense anxiety. When I was appointed in May we did not expect that the nation would soon be going through one of the most terrible times that it has had in all its history. I sincerely hope that by the time my presidency has come to an end, Britain will be in a happier, a stronger, and a nobler position than she has been in all recorded history.

The Council of the Society advised us to cut down the number of our meetings, as the other Sections have done, because so many men are away that the usual work of the Society will be very much interfered with. The dates of our three meetings will be duly made known.

With regard to our own special work, Otology, I would like to consider some of the problems in otology which have yet to be solved.

The problems in otology already solved by otologists are very considerable. If we look back twenty or thirty years and remember how little we knew and what a small amount we could do, even for a case of suppuration of the middle ear, to relieve patients from their ultimate fate, and when we see such cases now treated successfully, both by means of operation and without, we shall appreciate how much advance

has been made. We must consider how many lives have been saved, and how frequently hearing has been restored, or at all events improved. This certainly gives us hope for the future.

But we have now to consider what difficult problems lie before us. Take the cases unassociated with suppuration or with catarrhal conditions; how very misty the subject appears to us. We may first try to explain why it is that there is such a sharp line between those cases associated with catarrh or discharge on the one hand, in which we can forecast complications and do something to alleviate, and those cases, on the other hand, concerning which we are still very much in the dark. I think the explanation will be found to be this: With the discoveries of Pasteur and Lister of the influence of micro-organisms in producing disease the whole medical profession began at once to turn their energies into that channel. Bacteria received great attention, with the most wonderful results in all branches of medicine. Innumerable investigators sprang up to make a special study of this subject, and we know what remarkable consequences have followed. It has been said that there is the soul of good even in things evil. I submit that it is equally true that there is the soul of evil even in things good, because this extraordinary development of bacteriology entirely absorbed minds which might otherwise have devoted their attention to other aspects of pathology. Consequently, for many years the tissues and their manner of response to the invading bacteria were practically left out of consideration. The bacteria were considered as everything: the man as nothing. But, obviously, there cannot be disease without an animal—man or other. Disease, after all, is response to injury, whether by toxins of bacteria or other origin, or by chemical or physical agents. Therefore, the outstanding factor is not the organism or the poison, but the tissue that responds. If individuals were exact replicas of each other, then the same disease would always present exactly the same clinical phenomena. Cases of pneumonia would always have the same temperature, and the rise and the crisis would occur on the same day, the same hour. But no two individuals are alike! Consequently, when a poison or an injury affects the tissues we may give the condition a certain name, but it is never in the strict sense the same disease in all.

There are, however, a number of diseases which, apparently, can only affect certain individuals, whatever the environment may be; and in regard to these diseases we may say that the individual is by far the more important factor, the environment the less important. We may take, as an extreme example, Daltonism. No unfavourable environ-

ment will make the ordinary human being the subject of Daltonism. The condition must be innate, that is, inherited from parents or ancestors, or arising spontaneously. A much less extreme example, but still very noticeable, is arterio-sclerosis. No doubt the majority of people can be made to suffer from arterio-sclerosis; but a certain percentage of individuals might subject themselves to all kinds of unfavourable conditions, lead outrageous lives, and dissipate themselves in many ways, and yet live to a green old age! Others, though living careful and well-ordered lives, may suffer from arterio-sclerosis in comparative youth. A disease of which we see a good deal in otology is otosclerosis, and it is similar to arterio-sclerosis in this respect. It may appear in generation after generation of a family, though the victims may live in the most perfect surroundings, and with no apparent external cause acting at all. It seems as though in such individuals the tendency in the fertilised ovum is so strong that the disease will come into existence no matter what the environment may be. In spite of this, it remains true that in a large number of cases unfavourable circumstances are undoubtedly effective in producing otosclerosis. I am sure all of us must have seen cases of otosclerosis in which the exciting cause has been typhoid fever, prolonged anæmia in the form of chlorosis, pregnancy, and several other conditions. On the other hand, how very few people suffering from these general conditions become otosclerotics. It is obvious, therefore, that in otosclerosis the personal equation in large part determines the onset of the disease.

The question arises, why should that be? The tendency must be inherent, and must be passed down from parent to offspring. And yet if we try to arrange these cases of otosclerosis in accordance with the so-called Mendelian principle, we shall find that they do not group themselves in the way that colours of plants and heights of peas and beans do—characters which apparently do follow the Mendelian law. In otosclerosis we find the expected numbers are sometimes diminished, sometimes exceeded; and the same is true of arterio-sclerosis.

In regard to inherited neuroses, Dr. Mott has a very interesting paper in the *Archives of Neurology*,¹ and he came to the conclusion that these conditions cannot be brought into relationship with Mendel's law. And yet there is no doubt that the Mendelian law, generally speaking, is true in regard to certain characters. As far as I can make out, Mendel's law applies to those hereditary conditions which are

¹ *Arch. of Neurol. and Psych.*, 1914, vi, pp. 79-98.

part and parcel of the race or species. For example, Mendel mixed a pure strain of short peas with one of tall peas, and of the offspring some were tall, some short, but none medium. Shortness and tallness, however, are inherited qualities, and they are destined to appear from the beginning. The same is true of such a condition as night-blindness and Daltonism; they are, clearly, inherited conditions absolutely independent of any external agency or environment. Consequently, those oculists and biologists who have examined into the inheritance of night-blindness and Daltonism find that those conditions do come under the Mendelian law. But when we take arterio-sclerosis, otosclerosis, and the neuroses, we find that they do not come within that law. The reason, I venture to think, is not very difficult to find; it is that these pathological conditions are to a certain extent dependent on external environment; they are only *potentially* going to occur. The individual, with few exceptions, is not definitely destined to suffer from arterio-sclerosis; for a considerable number of the cases depend upon some unfavourable environment.

If we probe still further into this matter, we shall find that those diseases in which the condition only occurs when, in addition to an innate tendency, a certain environment is also necessary are characterised anatomically by the fact that the process of repair is deflected from its normal course. For instance, in the case of arterio-sclerosis, such a poison as lead introduced into the system will act on the arterial walls and cause injury. Consequently, certain cells will divide and multiply, probably a great many are destroyed, and some of the highly specialised cells, which have comparatively little power of reproduction, will be at a great disadvantage compared with those of the fibrous tissues, which have great capacity for reproduction. Hence the individual will begin to have an increase in the fibrous elements in the walls of his arteries, and a decrease in the more highly specialised muscular ones. Therefore, it is not surprising that such diseases do not manifest the Mendelian principle; they depend to a certain extent on some unfavourable environment. The individual who is liable to suffer from arterio-sclerosis and otosclerosis, because of his inherited tendency, needs also that other conditions should come into play before the disease will come into existence. Even malignant disease, if we take it locally, indicates to a certain slight extent an inherited tendency. But irritation of the part will establish the disease when it might not otherwise have shown itself.

My remarks, I fear, have been rather disjointed, but I cannot help

thinking that the line of future study, not of otology only, but of pathology in general, lies in a consideration of the tissue cells—as to why they undergo certain changes.

We talk of repair, but what is the biological significance of the process of repair? Taking the case of repair of a wound in the skin, we have a fair idea of *how* that process takes place. We know how the fibrous tissue cells divide, we know how the leucocytes come from the blood-vessels, how the epithelial cells grow and form a harmonious union of the connective tissue cells, and so on. We know that the epithelial cells change their character and become phagocytic, and the same is true of the fibrous tissue cells. That is to say, we have a fairly good knowledge as to *how* repair takes place. But, curiously enough, I have never, in all my reading and in all my communications with pathologists and physicians, heard the question asked, “*Why* does a wound heal?” Why, when we injure the tissues, does repair take place at all? That is the problem before pathology. Why does not the wound remain open as a raw surface?

When we solve the problem as to why repair occurs under normal conditions we may be in a position to say why it does not occur in certain other conditions, such as malignant disease, arterio-sclerosis, otosclerosis, and many more. We shall not solve the problems of pathology by knowing *how* repair is carried out, although that is extremely important; we shall have to know *why* it is carried out.

The problem will be difficult to solve, because no amount of observation of clinical facts or physiological or pathological experiments in laboratories or outside of them will suffice. What is required is the highest of all the intellectual qualities—imagination.

Brain of a Patient shown to the Section last year; the Patient having died some weeks after Operation.

By W. M. MOLLISON, M.C.

A TUMOUR is seen occupying the base of the brain mainly about the left cerebello-pontine angle: there is an extensive hæmorrhage into the growth, and occupying the left-hand side of the mass is a cystic cavity (opened at the operation). Microscopically the growth is a glioma.

DISCUSSION.

Mr. C. E. WEST thought the thanks of the Section were due to Mr. Mollison for his complete and painstaking account of a very interesting case. One was brought into occasional contact with these cases, and they were difficult of diagnosis, and treatment was a very formidable matter. This case was obviously impossible, surgically, at the time it was attacked. The point in the case which particularly interested him was the distension of the upper and lateral part of the fourth ventricle, simulating a cyst. Presumably it was cerebrospinal fluid, not cystic fluid in the sense of the contents of a cystic growth, which ran out of the wound. He doubted death from exhaustion if by that was meant exhaustion due to loss of cerebrospinal fluid, unless the patient showed signs of inadequate fluid in a general way, similar to those of continued hæmorrhage. He once nearly killed a patient by withdrawing a large quantity of cerebrospinal fluid, but she rallied and recovered, and, he thought, was still alive. He had withdrawn as much as 500 c.c. of that fluid in twenty-four hours without the patient showing signs of distress, and if the patient were kept well supplied with fluid he could stand great drains of his cerebrospinal fluid. Mr. Mollison generously presumed against himself that there was some infection of the meninges in this case, but the specimen did not show any grounds for that conclusion. Such patients as these died, and on the post-mortem table one could not say why, and that was particularly true of malignant cases. One case which he saw, but did not attempt to operate upon, had vertigo as the earliest symptom, and was pronounced inoperable by a very eminent cranial surgeon in London. After exploration he was subjected to X-rays through a decompression opening in the occipital region repeatedly. He was a student, and that was done two and a half years ago. Since then the patient had qualified.

Mr. G. J. JENKINS also desired to thank Mr. Mollison for the beautiful preparation and for his careful notes of the case. The point of interest to him, as it had been to Mr. West, was the association of a tumour and a cyst, and he asked whether Mr. Mollison could offer any explanation or suggestion to account for that association. It was curious that the whole of the fourth ventricle was dilated, and that the dilatation involved the left lateral recess of the ventricle—in the region of the cornucopia. It was possible that this recess had formed the cyst. It was difficult to see the anatomical formation in the specimen, but the association of cyst and tumour was interesting. Might there be a section of the lining membrane of the cyst? as it would be important to know its histology in coming to a conclusion as to the nature of the cyst.

Mr. MOLLISON replied that Dr. Wyatt Wingrave told him before the discussion that he had a similar case which was a papilloma of the

choroidal plexus, and that microscopically it was either an endothelioma or an epithelioma. In the light of that knowledge, perhaps one might change one's diagnosis in this case to endothelioma; though the microscopical difference between the two was a very subtle one. Dr. Johnson, who had specialised in the pathology of the brain, reported it was a glioma, with neuroglial elements in it. He would read Dr. Johnson's report: "Histologically the tumour exhibits the structure of a glioma. In parts the section shows typical neuroglial formation. The ground substance consists of a delicate protoplasmic network formed by the branching processes of the tumour cells. The nuclei of these latter are large and well defined, and are surrounded by a thin layer of protoplasm. Occasionally small areas of degeneration occur, which appear to be early collections of fluid between the cells. In other places, possibly the more rapidly growing portions of the tumour, the cells are more closely packed and the ground substance is proportionately less. Here the appearance approximates somewhat to a sarcomatous condition. This tendency, however, is insufficient to justify the application of the term 'glio-sarcoma' to the specimen." Dr. Johnson mentioned that there was some degeneration, with fluid between the cells, so a possible explanation of the cyst would be that it was an extension of that process over a large area. Mr. Jenkins made to him an excellent suggestion for the presence of the fluid, namely, that there was some nipping by the growth of the choroidal plexus, and consequently a kind of œdema behind. Dr. Johnson was now away at the war, but as soon as he knew anything further he would communicate it.

Case of Syphilitic Ulceration of External Auditory Meatus.

By W. M. MOLLISON, M.C.

W. C., AGED 32, has been attending the Out-patient Department at Guy's Hospital, suffering from a chancre. This has been treated by means of mercurial injections. Six weeks ago a boil appeared in the left ear; this has been discharging ever since. The external auditory meatus and tragus on the left side are the seat of an ulceration. The meatus is so filled with flabby granulations that the membrane cannot be seen. There seems to be little or no pain in connexion with the ulceration. The soft palate shows a condition of superficial ulceration.

Slides illustrating Lumbar Puncture Fluid in Aural Cases.

Shown by WYATT WINGRAVE, M.D.

CEREBROSPINAL infection in *chronic* aural cases is usually polymicrobial; in *acute* cases monomicrobial. Lumbar puncture for first or even second "tap" is often negative, due partly to localisation or to slow circulation of fluid.

Chemical and physical characters are also profoundly affected; their significance consists in absence of sugar, presence of acid, increase in specific gravity.

Early appearance of cytological changes in labyrinthine complications.

The PRESIDENT (Dr. Albert A. Gray) said the Section was very much indebted to Dr. Wingrave for his exhibit and demonstration. He (the speaker) did not feel competent to discuss it; he was in the habit of sending the results of his punctures to the pathologist and the chemist.

Case of Carcinoma of the Pinna.

By C. E. WEST, F.R.C.S.

W. A., AGED 69, male. Seven years ago patient had a hard wart on the left cheek. This was excised, but returned quickly. It was destroyed by carbon dioxide snow and has not returned. For several years he has had a warty growth on the dorsum of the wrist, and lately a small papilloma at the inner canthus of the right eye. Eighteen months ago a small ulcer appeared on the left ear; this has rapidly increased lately. Patient has not used tobacco and alcohol for thirty-four years. Blood gives a negative Wassermann reaction.

On admission to hospital, September 4, 1914, there was a large ulcerated septic growth involving the inner aspect of the tragus, the external meatus and concha on the left side. The tissues on the posterior aspect of the pinna and over the mastoid region were oedematous and brawny, and one or two small glands could be felt in the upper part of the anterior triangle of the neck.

Operation, September 9: A racket-shaped incision was carried round the pinna, including the area of the mastoid and about three-quarters of an inch of the skin of the cheek. The skin was raised anteriorly up to the margin of the parotid, and the superficial tissues were dissected from the gland with the parotid fascia backwards to the meatus. Above, the tissues were raised down to the temporal fascia, and behind down to the bone. When the meatus was reached, this was separated from the bone, and the pinna removed with the surrounding tissues *en bloc*. An extensive radical mastoid operation was now carried out, with a wide exposure of dura mater above and the removal of the whole of the bony walls of the meatus except a small part of the deepest portion of the floor. The facial nerve was not exposed in this part of the operation. The handle of the racket was now lengthened for some 3 in. over the sternomastoid, the muscle was detached from the bone, turned down, and transfixed and ligatured and cut away. Beginning from the under surface of the petrous, the substernomastoid tissues were dissected off the carotid and jugular, and as far forward as the attachment of the stylomandibular ligament and posteriorly from the apex of the posterior triangle, and this dissection was carried down as far as the level of the cricoid cartilage, in a continuous sheet. The neck wound was closed and drained at the lower end.

Some branches of the facial nerve were known to have been divided at their exit from the parotid, and it is probable that the trunk of the nerve was injured in the deep dissection on the under surface of the petrous.

The wound granulated well, the edges rapidly drawing in, and on October 21 the cavity was lightly curetted under cocaine and adrenalin and a graft applied. There was at first a considerable diffuse induration round the cut end of the sternomastoid, but this has gradually diminished.

The case is shown as an illustration of the removal of a primary growth with its surrounding area and lymphatic territory in continuity. The amputated pinna and a section of one of the glands in the mass of tissue from the neck are exhibited.

DISCUSSION.

Mr. WEST added that, as he said in the notes, the case was shown to illustrate the removal of a primary growth with its surrounding area and lymphatic territory in continuity. Both the partial success and the ultimate failure hinged upon that. He ought further to say that the man had that

afternoon been demonstrated to have palpable glands above the left clavicle. The ideal thing to have done in such a case was to do a complete dissection beneath the sternomastoid and in the whole of the posterior triangle at the original operation, and as this patient showed very little bad effect from the operation, he believed he would have stood that being done. But the difficulty would be to get him up to the operating table a second time, even though he was so grateful for the relief which had been given him. It illustrated how wide an area ought to be included in the operation in these cases, either all at the primary operation or as an immediate sequel, in order to secure freedom from recurrence.

Mr. G. J. JENKINS considered that Mr. West was to be congratulated. The result was a remarkably fine one for such a condition. The patient was of the type in which recurrences were apt to take place; the multiple papillomata indicated his cancerous tendency. In infirmaries these people were looked upon as likely subjects for cancer. This patient had had papillomata, some of which had been cured by radium. He would like Mr. West's opinion on this point.

The PRESIDENT wished to join in the congratulation expressed by Mr. Jenkins. Even though recurrence might take place, and indeed probably would, the great point was that the terrible condition had been obviated which ensued from ulceration about the meatus, which involved the bone. Even if he had recurrence in the glands near the clavicle, he would not suffer as he would have done had the operation not been undertaken. The way in which the grafting succeeded was particularly fortunate, and showed how thoroughly the malignant tissue must have been eradicated.

Mr. C. E. WEST, in reply, thanked those who had spoken so kindly about the case. But he confessed that, personally, he fought malignant disease to win, not merely to delay death; consequently he was disappointed at the signs of recurrence. He agreed with Mr. Jenkins that this patient was a typical example of the type of person who developed squamous carcinoma in some part, and one who was likely to have a local recurrence. But he did not feel that this absolved him from the disappointment that there should be a recurrence in the glands, because he did not think glandular recurrence had a bearing on the question of the liability of a new primary epithelioma developing if the patient should live long enough.

A Cold-air Labyrinth Testing Apparatus.

Shown by J. DUNDAS GRANT, M.D.

THE apparatus consists of a metal tube covered with an absorbent linen webbing and coiled in the form of a hollow cone. The free



FIG. 1.

Cold-air labyrinth testing apparatus.

extremities of the tube are insulated by means of rubber tubing of which a portion projects beyond the metal for insertion in the ear: the distal part of this is separated from the rest and can be easily

changed to meet æsthetic or aseptic requirements. The absorbent covering is well wetted with chloride of ethyl and air is then blown through the tube by means of an ordinary spray bellows. In order to make the flow of air gentle and continuous it was found necessary to use a fairly strong secondary bag to the bellows and to choke very considerably the bore of the proximal end (of the metal tube).

When the air blown on the cheek is felt to have reached a high degree of coldness the tube is introduced into the meatus of the ear so that the cold blast impinges directly on the tympanic membrane. Inflation is continued until nystagmus is or ought to be induced.



FIG. 2.

Cold-air labyrinth testing apparatus. Method of application.

When the instrument is thus arranged the maximum of coldness is obtainable in from twelve to fifteen seconds and is kept up for about seventy-five seconds, when more chloride of ethyl can be added easily. In most normal cases the nystagmus is induced in an average of thirty seconds, the head being thrown back 60° beyond the vertical and the eyes being intermittently directed towards the opposite side at an angle of 50° from the middle line (Brünings's optimum position) and up to the ceiling.

DISCUSSION.

The PRESIDENT asked whether there was any possibility of doing harm to the membrane by such an apparatus. The use of ether, of course, would bring the air down almost to freezing temperature. He supposed that when it was put into practice the test would be standardised.

Dr. URBAN PRITCHARD referred to a case in which it was intended to incise the membrane, but the surgeon thought he would try local anæsthesia. He sprayed in the ether, and the patient dropped to the floor immediately, and the process had to be stopped.

Mr. O'MALLEY said that three years ago, in Vienna, he had an opportunity of using a somewhat similar apparatus, which was devised by Bárány. It had the usual spraying apparatus, with a double-channel tube passing into a bottle which had some ether at the bottom. But this apparatus of Dr. Grant's seemed to be a considerable improvement on Bárány's, because it only impinged cold air on the meatal wall, whereas in Bárány's apparatus ether vapour accompanied the cold air, and he did not think it was wise to introduce a drug in vaporised form into the ear. Bárány devised it for use in cases of perforation of the tympanic membrane in which he did not like to use cold water, for fear of setting up otitis media. He questioned the advisability of introducing sudden cold to the membrane, and through that to the labyrinth.

Dr. JOBSON HORNE considered it would be advisable to know the effects of ethyl chloride in a cold-air labyrinth testing apparatus upon the normal subject before gauging its clinical value in disease. Doubtless time would show that the instrument had its usefulness and also its limitations and contra-indications.

Mr. E. D. DAVIS asked Dr. Grant if his apparatus could be modified for the use of hot air in addition to cold. It was suggested that a movable chamber for hot water could be placed within the spiral tube.

Dr. DUNDAS GRANT replied that he had seen no harm result from the use of the apparatus. He could conceive such occurring as the result of spraying ether into the ear, as had been suggested by some writers. The lowest temperature he had obtained, tested by a thermometer, was $44\frac{6}{10}^{\circ}$ F., or $7\frac{1}{4}^{\circ}$ C., which was still considerably above freezing temperature. In reply to Dr. Jobson Horne, nystagmus, in Brünings's position, was caused in about twenty-eight seconds after the apparatus had been well cooled. After compression during ten or twelve seconds it was found to be quite cold to the cheek. In about twenty-eight to thirty-two seconds later (the time at which nystagmus appeared normally to occur) the temperature was down to about 48° F. He agreed it might advisably be standardised in detail, and he had tabulated the rise and fall as observed at intervals of five seconds. The idea of using it also

for heated air had occurred to him, but he had as yet done nothing to carry the idea out. Perhaps a small lamp could be used, or a mixture of sulphuric acid and water, to produce the necessary heat. An electrically heated platinum wire could be employed, as in Wylie's apparatus, for passing hot air into the Eustachian tube, but this was not portable. The essential feature of the apparatus exhibited was its portability and convenience.

Dr. VINRACE proposed a cordial vote of thanks to the President for the valuable address he had delivered and for his kindness in coming so far as from Glasgow to preside over the meetings of the Section. The address gave a very broad and enlightened account of the ætiology of disease, and he was very pleased that the President fully recognised that the throat, nose, and ear ought not to be considered by themselves. The address dealt in detail with questions of heredity, and the need for giving attention to the processes of gestation had been enforced in a stately and dignified way. Too often such addresses dealt with a very circumscribed region, and lacked a broad consideration of disease as a whole.

The resolution was carried.

The PRESIDENT, in thanking Dr. Vinrace and the Section for the kind resolution, said he thoroughly enjoyed meeting his colleagues here in London; he had always considered his visits to the Section well worth the expenditure of time involved, and he intended to continue them. With regard to the subject of the address itself, we had a long way to go before the problem of the response of the tissues to injury was settled. It was a biological problem, and pathology was, after all, only a branch of biology.

Otological Section.

February 19, 1915.

Dr. ALBERT A. GRAY, President of the Section, in the Chair.

DEMONSTRATION OF CASES AND SPECIMENS ILLUSTRATING TUBERCULOSIS OF THE AUDITORY APPARATUS, FOLLOWED BY GENERAL DISCUSSION (p. 35).

Tuberculosis of the Ear.

Specimens exhibited by WYATT WINGRAVE, M.D.

- (1) Tubercle bacilli in discharge. Stained by picro-fuchsin method.
- (2) Acid-fast bacilli in discharge, simulating *Bacillus tuberculosis*.
- (3) Acid-fast bacilli in atrophic rhinitis.
- (4) Giant cells of tuberculoma.
- (5) Giant cells of non-tubercular granuloma.

The Clinical Aspect of Tubercular Disease of the Ear.

By A. LOGAN TURNER, M.D.

FIFTY-ONE children, during a period of eight years, under the age of 15, with discharge from one or both ears, in which there was evidence, direct or indirect, to lead to the conclusion that the middle-ear disease was *tuberculous*. During same period, aged under 15, there were 1,797 cases of middle-ear suppuration, therefore 2 per cent. were *tuberculous*. As 48 of the tuberculous cases were aged under 5, and there were 505

cases of middle-ear suppuration under that age, 9 per cent. were tuberculous. In 43, or 84 per cent., disease commenced in the first year of life. (Nine adults showed evidence of tubercle.)

Sex.—Males, 26; females, 25; total, 51.

Ear.—Right ear only, 26; left ear only, 14; bilateral, 11; total, 51.

Mode of Onset.—92 per cent. of quiescent origin, pain being a conspicuously absent phenomenon.

First Clinical Sign noticed.—In 45, or 88 per cent., the discharge; in 2, the glands; in 1, discharge and facial paralysis; in 3, no note.

Ætiology.—(a) Method of feeding; (b) other evidence of tuberculosis. (a) *Feeding*: Bottle-fed, 30, or 88 per cent. of 34 stated; breast-fed, 4, or 11 per cent; not stated, 17. (b) *Other evidence of tuberculosis*: Enlargement of periotic lymph glands an outstanding feature in 42 of 44 cases stated—i.e., 95 per cent.

The question of tuberculosis in the child's family, or elsewhere in the child, was not as a rule inquired into in a thorough manner. There are a few isolated facts: In three cases the mother was dying of pulmonary tuberculosis; in one case the father had pulmonary tuberculosis; in one case a brother had Pott's disease; in one case a twin had ear disease and tuberculous meningitis; in one case the child had pulmonary tuberculosis; in one case there was lupus of soft palate and pharynx.

Facial Paralysis.—Present in 23, or 45 per cent.

Concomitant Affection of the Labyrinth.—Approximately determined in the 35 cases operated upon, labyrinth destroyed in whole or in part in 8, or 22 per cent. The outer labyrinth wall showed changes in 11 others—e.g., erosion, pitting, softening of bone, and some of these may later have developed destruction of labyrinth.

Tuberculous Meningitis.—Four died of tuberculous meningitis—i.e., 7 per cent.—two after the mastoid operation, two without operation.

Pathological Findings in the 35 Cases operated upon.—Definite evidence of tubercle sought for in 17 cases—1, inoculation of guinea-pig and development of general tuberculosis; 2, tubercle bacilli in the ear discharge; 14, giant cell systems and caseation in the granulations (in one of these tuberculous adenoids). Sequestra (45 per cent.), carious bone (48 per cent.), granulations, and caseous material common. In no case was cholesteatoma found.

Tubercular Disease of the Ear (Pathology).

By J. S. FRASER, M.B.

(I) CASES.

Case I.—W. Y., aged 1 year 3 months. Child was bottle-fed. Parents noted one month ago that the child's face was a little drawn to the right side. No history of measles or fever, but both ears have been discharging for some months; apparently child has never had any pain in the ear.

Examination: Partial paralysis of left side of face. Foul pus in left meatus and swelling of posterior wall. No mastoid swelling, but a few shotty glands behind sternomastoid. Right meatus also contains pus and granulation tissue.

June 4, 1913: Operation, left side. Cortex healthy, antrum contained granulation tissue, bone around soft; Eustachian tube contained swollen mucosa; granulations in oval and round windows; large area of dura exposed to get beyond diseased bone. Malleus and incus absent; no naked-eye appearance of labyrinth disease.

June 9, 1913: Stitches removed; offensive discharge. Temperature normal before and since operation.

June 20, 1913: Left ear clean, but right very dirty.

June 26, 1913: Operation right ear. Bone rather soft; a good deal of granulation; inner wall seemed healthy.

June 27, 1913: Temperature rose to 102.6° F.

June 28, 1913: Child very restless, has a dusky colour this morning; rapid respiration; squinting; vomiting; broncho-pneumonia present. 4 p.m.: Temperature, 104° F. Child died in the evening.

Post mortem: Tuberculosis of right lung and of bronchial glands. Mesenteric glands enlarged and caseous. No tubercle of brain.

Note.—Guinea-pigs inoculated from lymphatic glands removed from back of ear showed definite tuberculosis. Granulation tissue from the ears showed small tubercular areas. The photomicrographs were prepared from the right, in which the tubercular disease was less advanced than in the right. The illustrations show a comparatively early stage of tubercular disease of the ear. The labyrinthine involvement through the oval and round windows is just beginning.

The photomicrographs were prepared from the right ear, in which

tubercular disease was less advanced than in the left. The illustrations show a comparatively early stage of caseous tubercular disease involving the mucous membrane of the middle ear and eroding the bony wall of the promontory. The labyrinthine involvement through the oval and round windows is just beginning (*see* figs 1, 2, 3, 4 and 5).

Case II.—J. N., male, aged 9 months (a twin), had epileptic fits at the age of 6 weeks; a fortnight later the right ear began to discharge; the fits continued at intervals. At the age of 3 months the right ear was operated upon at another hospital, but the discharge continued.

Examination (August 3, 1910): Child markedly wasted; mouth shows thrush; frequent vomiting; profuse nasal discharge (*Staphylococcus aureus*, diphtheroid bacilli and diplococci). Child's head sweats profusely and is markedly retracted; photophobia present. There are enlarged glands below the right ear, and behind the ear there is a fistula discharging pus. The right side of the face is paralysed.

August 4, 1910: Operation. Large flat sequestrum removed from roof of middle ear, exposing the dura. Sequestrum also removed from floor of middle ear and another from the inner wall, opening up the cochlea, vestibule, and canals.

August 5 to 16, 1910: Progressive emaciation and glandular enlargement; wound cavity very dirty and shows few granulations.

August 17, 1910: Death.

Post mortem: Membranes of brain injected; increase of cerebrospinal fluid. Tubercular nodule in right anterior corpus quadrigeminum and another in the occipital region. The dura of the right middle fossa showed tubercular thickening.

The photomicrographs show advanced tubercular disease of the ear. There has been extensive necrosis of the outer wall of the vestibule and also in the region of the semicircular canals. The Eustachian tube is not recognisable, and the tubercular process has reached the wall of the internal carotid artery (*see* figs. 6, 7, 8, and 9).

Case III.—J. C., male, aged 9 months (breast-fed). Nasal catarrh and obstruction since birth; left side otorrhœa of ten weeks' duration; no history of crying before discharge began; swelling behind left ear for eight weeks. Wilde's incision made by patient's doctor.

Examination: Very marked adenoids; large kidney-shaped perforation of left middle turbinal. Otorrhœa right side also.

Adenoid operation: Child admitted to ward; vomiting after meals; no signs of tubercle bacilli in lungs.

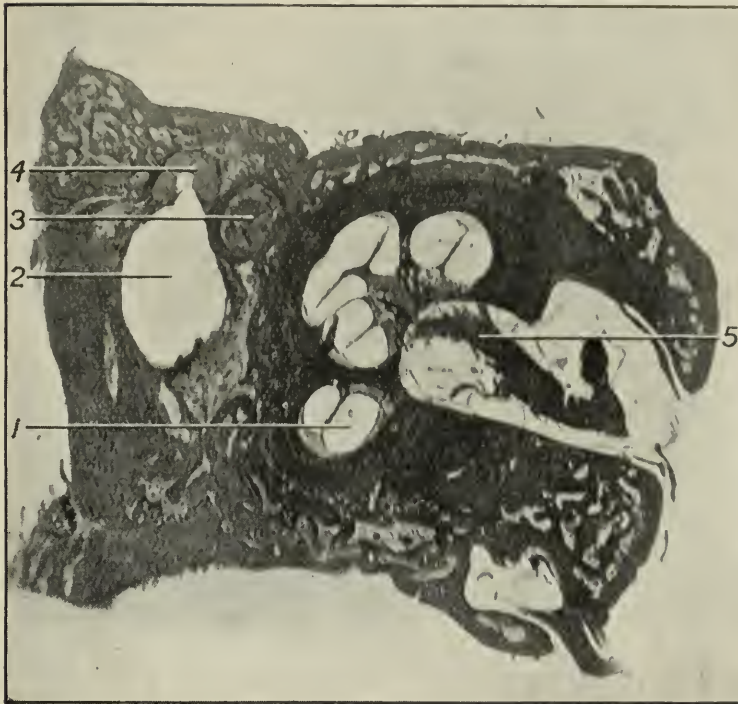


FIG. 1.—*Case I*: W. Y., aged 1 year 3 months. Early tubercular disease of the ear (right ear, section 140). Inner wall of antrum. Tubercular infiltration and erosion of walls of tympanic cavity. 1, basal coil of cochlea; 2, tubal portion of tympanic cavity; 3, tensor tympani; 4, tubercular tissue in roof of cavity; 5, cochlear nerve in internal auditory meatus.

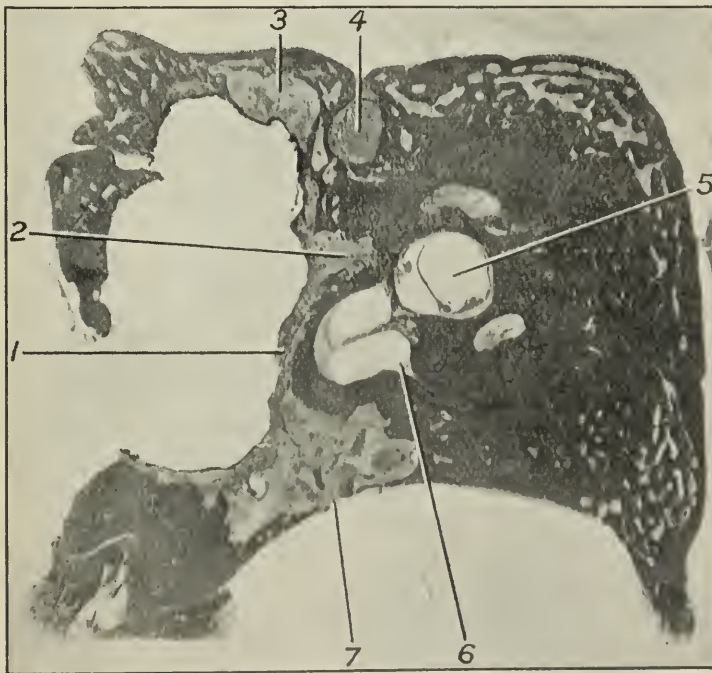


FIG. 2.—*Case I*: Early tubercular disease of the ear (right ear, section 305). Showing tubercular infiltration of floor of tympanic cavity reaching the jugular bulb. 1, tubercular erosion of promontory; 2, tubercular tissue in niche of oval window; 3, tubercular tissue in roof of tympanum; 4, facial nerve; 5, sacculus; 6, cochlear opening of ductus perilymphaticus; 7, tubercular granulation tissue extending down to jugular bulb.



FIG. 3.—*Case I*: Early tubercular disease of the ear (right ear, section 350). Showing invasion of vestibule through oval window; erosion of promontory; niche of round window filled with tubercular granulation tissue. 1, membrane of round window; 2, tubercular erosion of promontory; 3, head of stapes; 4, tubercular tissue in niche of oval window; 5, facial nerve; 6, vestibular nerve to utricle, external and superior canals; 7, footplate of stapes, eroded and displaced towards vestibule; 8, vestibular nerve to ampulla of posterior canal; 9, tubercular tissue filling up niche of round window



FIG. 4.—*Case I*: Early tubercular disease of the ear (right ear, section 425). Showing erosion of inner wall of tympanum. 1, tubercular tissue in region of sinus tympani; 2, footplate of stapes displaced towards vestibule; 3, ampullary end of superior canal with crista and cupula, the latter surrounded by coagulated lymph; 4, cranial end of fossa subarcuata; 5, endolymphatic space of crus commune; 6, crista quarta; 7, ampullary end of posterior canal; 8, jugular bulb.

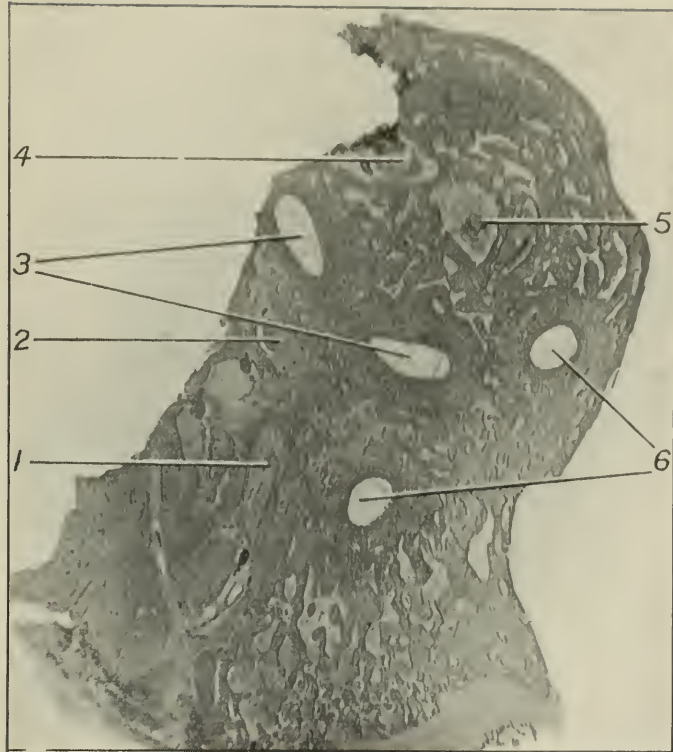


FIG. 5.—*Case I*: Early tubercular disease of the ear (right ear, section 600). Inner wall of antrum. Showing path of invasion from inner wall of antrum along vessels of fossa subarcuata. 1, stapedius muscle; 2, facial nerve; 3, two ends of external canal; 4, tubercular tissue on inner wall of aditus; 5, tubercular infiltration in fossa subarcuata; 6, two ends of posterior canal



FIG. 6.—*Case II*: J. N., aged 9 months. Advanced tubercular disease of the ear (section 90). Vertical transverse section through apex of petrous bone. 1, region of Eustachian tube; the cartilaginous tube has disappeared; cascating tubercle is seen to the left of and below the internal carotid artery; 2, internal carotid; 3, apex of petrous bone; in this region the bone consists largely of marrow spaces.

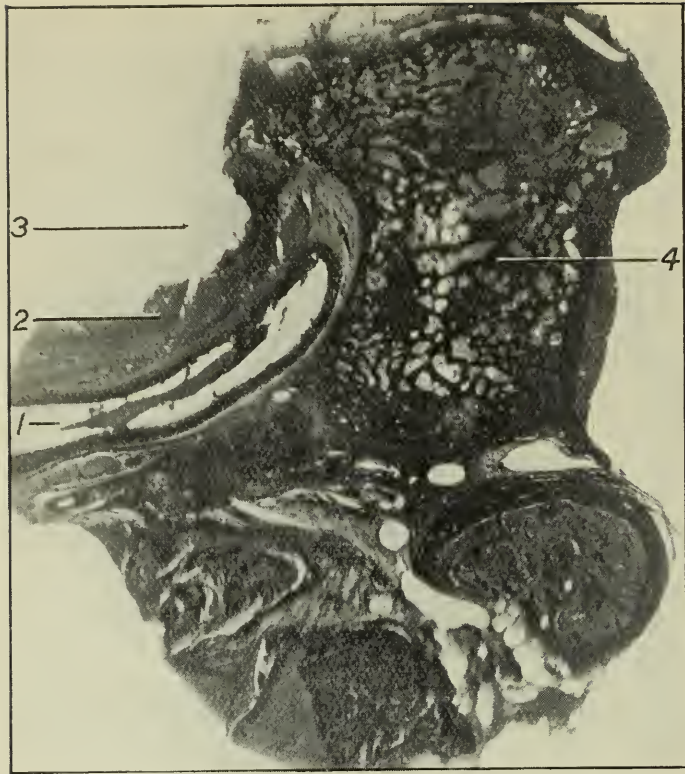


FIG. 7.—*Case II*: Advanced tubercular disease of the ear (section 200). Vertical transverse section through anterior part of petrous pyramid. 1, internal carotid artery; 2, tubercular granulation tissue infiltrating the wall of carotid artery; 3, region of Eustachian tube; the tube can no longer be recognised; 4, anterior part of petrous pyramid.

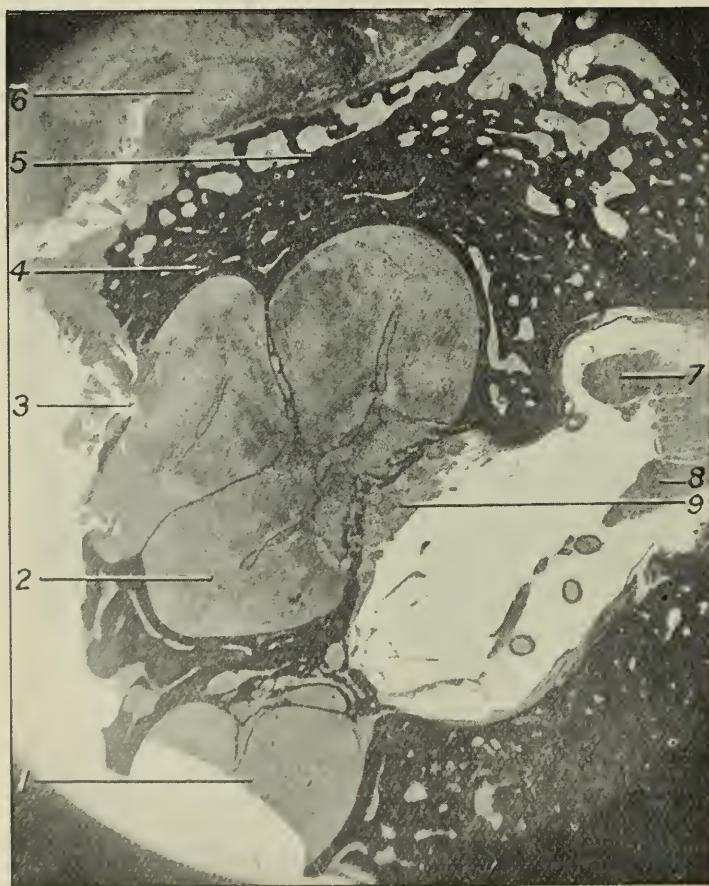


FIG. 8.—*Case II*: Advanced tubercular disease of the ear (section 337). Vertical transverse section through cochlea, showing fistulae into cochlea; cochlea is full of tubercular granulation tissue. 1, fistula into basal coil of cochlea; 2, middle coil of cochlea; all scalæ are filled with tubercular granulation tissue; the osseous spiral lamina is still to be seen; 3, fistula into apex of cochlea; 4, cartilage bone capsule of cochlea; 5, lamellar bone surrounding cochlear capsule; 6, tubercular pachymeningitis in floor of middle cranial fossa; 7, facial nerve; 8, auditory nerve; 9, cellular infiltration in fundus of internal meatus.

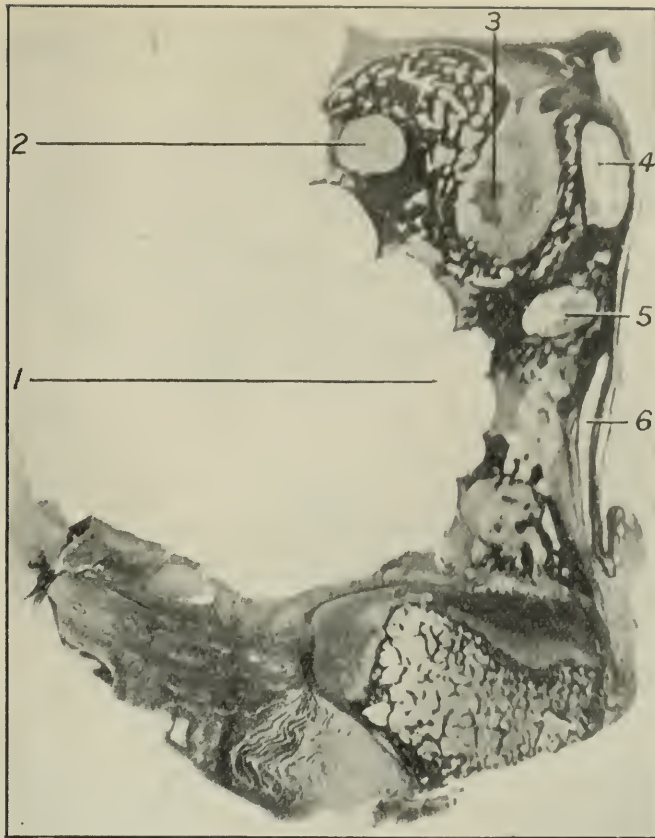


FIG. 9.—*Case II*: Advanced tubercular disease of the ear (section 750). Vertical section through posterior part of petrous bone. The outer wall of the vestibule has been destroyed by the disease. 1, large gap in wall of labyrinth; the ampullary end of the posterior canal, both parts of the external canal and the facial nerve are absent; 2, ampullary end of superior canal; 3, tubercular infiltration in fossa subarcuata; 4, smooth end of superior canal; 5, smooth end of posterior canal; 6, saccus endolymphaticus; the tubercular infiltration reaches to the outer wall of the saccus.

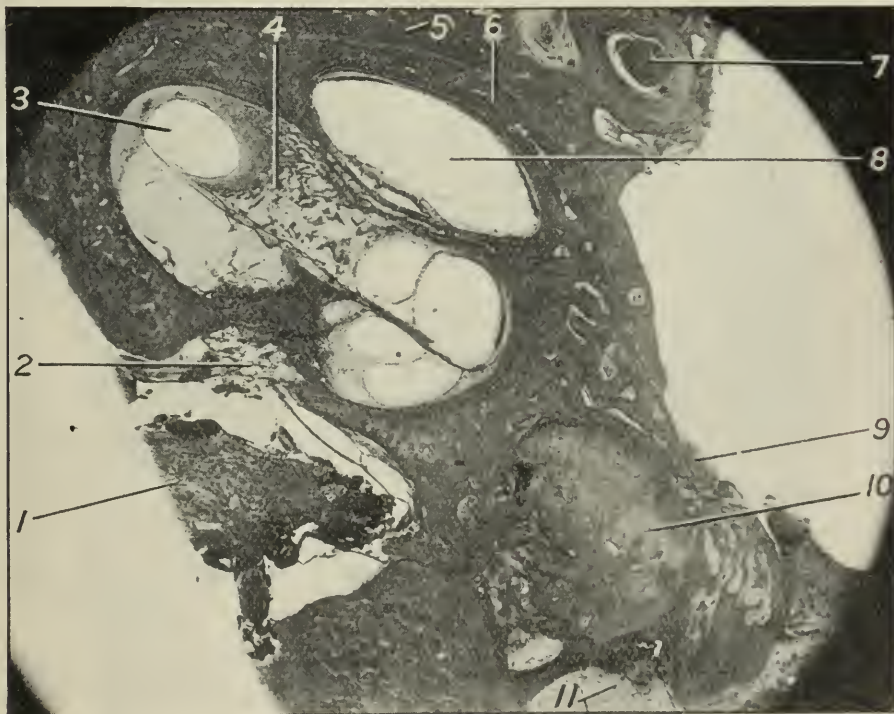


FIG. 10.—*Case III*: J. C., aged 9 months. Fibro-ossifying type of tubercular otitis. Vertical section of left inner ear (section A). 1, cochlear nerve in internal meatus infiltrated by tubercle; 2, small cell infiltration in fundus of internal meatus; 3, dilatation of cochlear canal in middle coil; 4, new formation of fibrous tissue and bone in scala vestibuli of middle coil; 5, lamellar bone surrounding cochlear capsule; 6, cartilage bone capsule of cochlea; 7, facial nerve; 8, apical coil of cochlea; 9, tubercular erosion (fistula) into basal coil; 10, basal coil filled by granulation tissue and new bone; 11, niche of round window filled with tubercular granulation tissue.

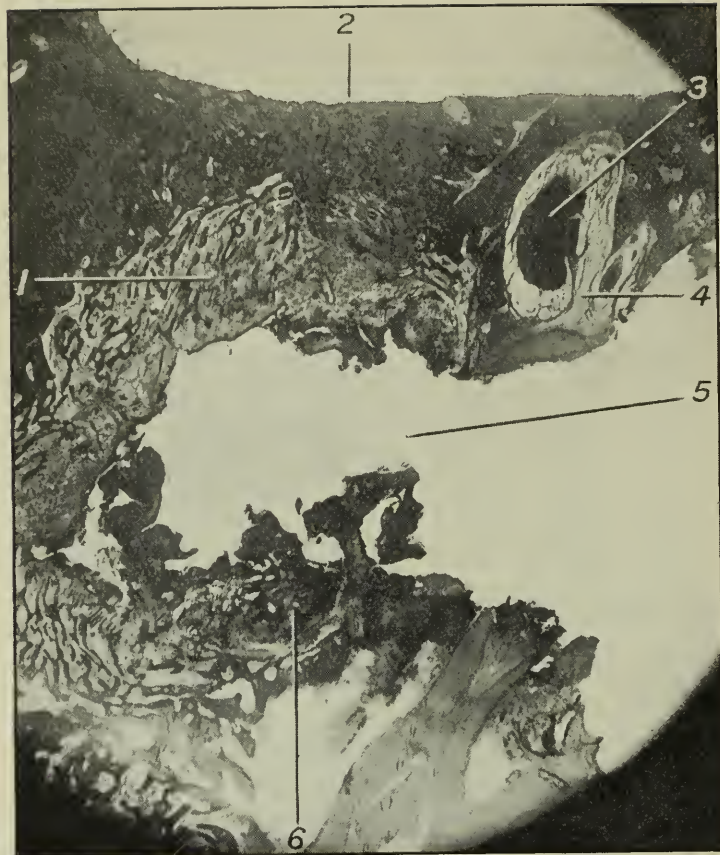


FIG. 11.—*Case III*: Fibro-ossifying type of tubercular otitis. Vertical section of left inner ear (section B). 1, new connective tissue and bone in wall of vestibule; 2, middle cranial fossa; 3, facial nerve; 4, dehiscence in facial canal; 5, large fistula into vestibule in region of oval window—the promontory has disappeared; 6, tubercular sequestrum in lower part of vestibule.

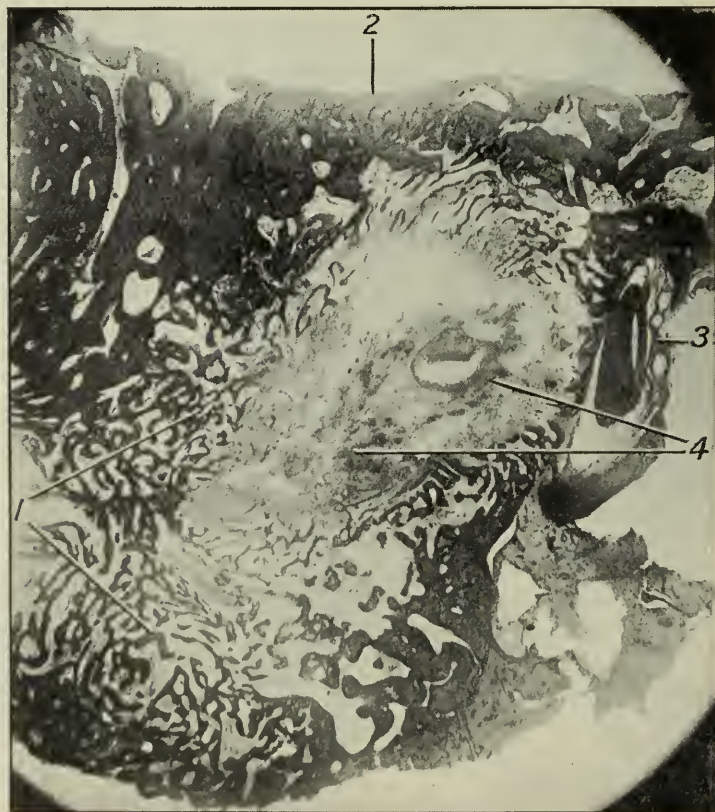


FIG. 12.—*Case III*: Fibro-ossifying type of tubercular otitis. Vertical section of left inner ear (section C). 1, new formed bone partially filling up vestibule and region of ampullary end of posterior canal; 2, middle cranial fossa; 3, dehiscence in facial canal caused by (?) tubercular erosion; 4, tubercular granulation tissue in centre of vestibule.

Radical mastoid operation : Much granulation tissue.

Progress : Vomiting continued ; head retraction developed ; meningitic cry ; internal strabismus left eye ; no optic neuritis or tubercle of choroid ; cerebrospinal fluid clear—slight increase in mononuclear cells. Death.

Post mortem : Tubercular meningitis and general tuberculosis.

The photomicrographs show a fibro-ossifying type of tubercular disease. To the best of the writer's knowledge, this type has not hitherto been described in connexion with tuberculosis of the ear, though it is well recognised in tuberculosis of the long bones. As will be seen from figs. 10, 11, and 12, there is considerable tendency towards spontaneous cure of labyrinthitis by the formation of granulation and fibrous tissue in the cochlea and vestibule, and its subsequent conversion into new bone. In this case the invasion appears to have taken place mainly through the oval window into the vestibule, and, to a less extent, through the round window into the cochlea (*see* figs. 10, 11, and 12).

(II) EXPERIMENTAL TUBERCULAR DISEASES OF THE EAR.

In 1910 and 1911 the writer performed fourteen experiments on guinea-pigs. The animals were anæsthetised and the right tympanic cavity was inoculated through the tympanic membrane with emulsions of various organisms. The experiments may be divided into two groups : (a) In nine cases, in which the tubercle bacillus was *not* injected, the organisms employed were as follows : *Staphylococcus aureus*, 1 ; *Streptococcus pyogenes*, 2 ; pneumococcus, 1 ; *Bacillus coli*, 2 ; *Bacillus proteus*, 1 ; *Bacillus of distemper*, 2. In only four out of the nine cases was otitis media found to be present in the inoculated tympanic cavity at the post-mortem, and in no case was labyrinth suppuration discovered on subsequent microscopic examination of the ear. (b) In five guinea-pigs the tubercle bacillus was employed for inoculation—in four cases in pure culture and in one in combination with the *Staphylococcus aureus*. In only one of the five cases was there failure to produce otitis media ; in one case there was otitis media and slight serous labyrinthitis (tubercle bacilli) present in the middle-ear pus. In the other three cases (guinea-pigs *D*, *N*, and *P*) there was tubercular otitis media and labyrinthitis—the inner ear being invaded through the oval and round windows (*see* figs. 14 to 20).

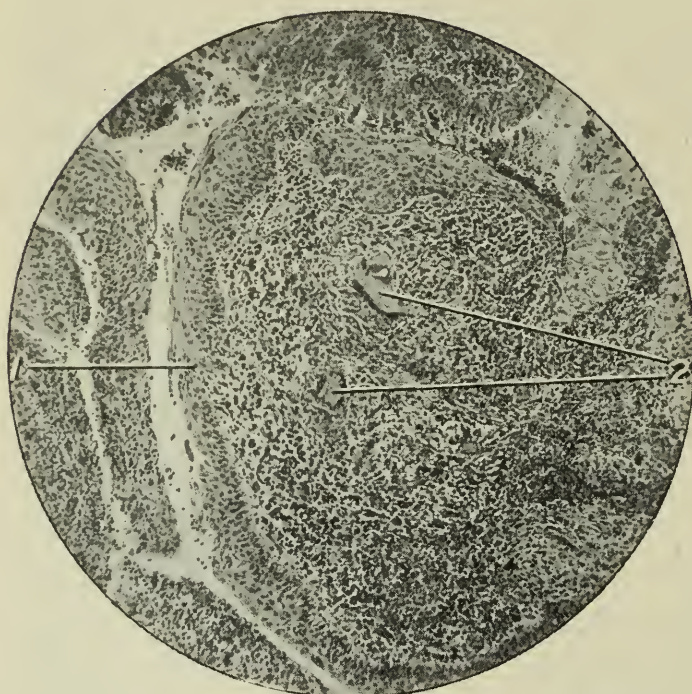


FIG. 13.—Tubercular disease of adenoid post-nasal growths. 1, stratified squamous epithelium on surface of adenoid growths replacing the normal stratified ciliated columnar epithelium; 2, giant cells in subepithelial lymphoid tissue.

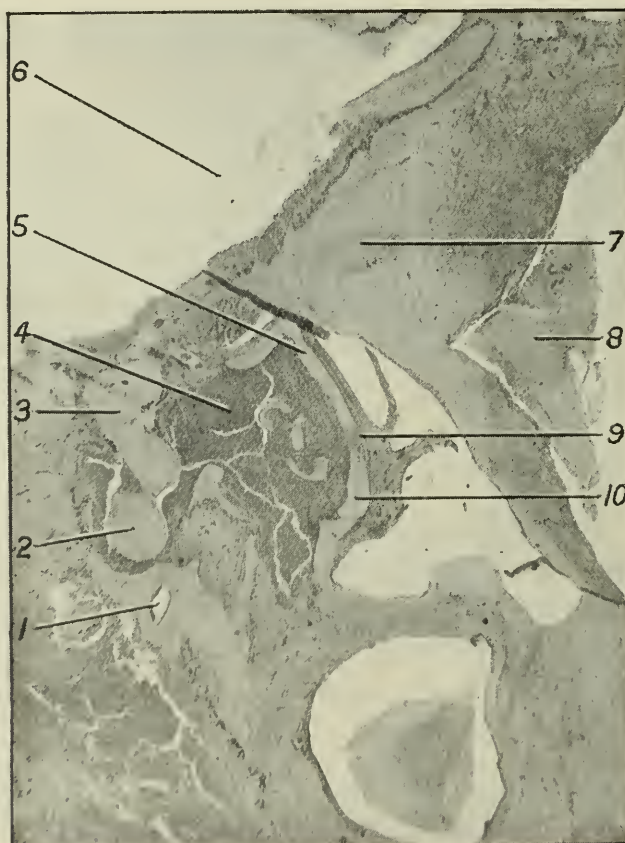


FIG. 14.—*Guinea-pig P*: Tubercular disease of the ear (experimental) (section 85). Horizontal section through right ear, showing perforation of footplate of stapes by pus and invasion of perilymph space of vestibule. 1, external canal; 2, incus; 3, malleus; 4, pus in tympanic cavity; 5, anterior part of footplate of stapes; 6, external meatus; 7, capsule of cochlea; 8, nerves in internal meatus; 9, perforation of footplate of stapes by pus, which is invading the perilymph space of the vestibule; 10, posterior part of stapes footplate.



FIG. 15. — *Guinea-pig P*: Tubercular disease of the ear (experimental) (section 105). Horizontal section through right ear, showing invasion of perilymph space of basal cochlear coil through round window. 1, external canal; 2, pus in niche of round window invading scala tympani of basal coil; 3, new connective tissue in scala tympani of basal coil of cochlea; 4, vestibule; 5, sinus tympani full of pus.

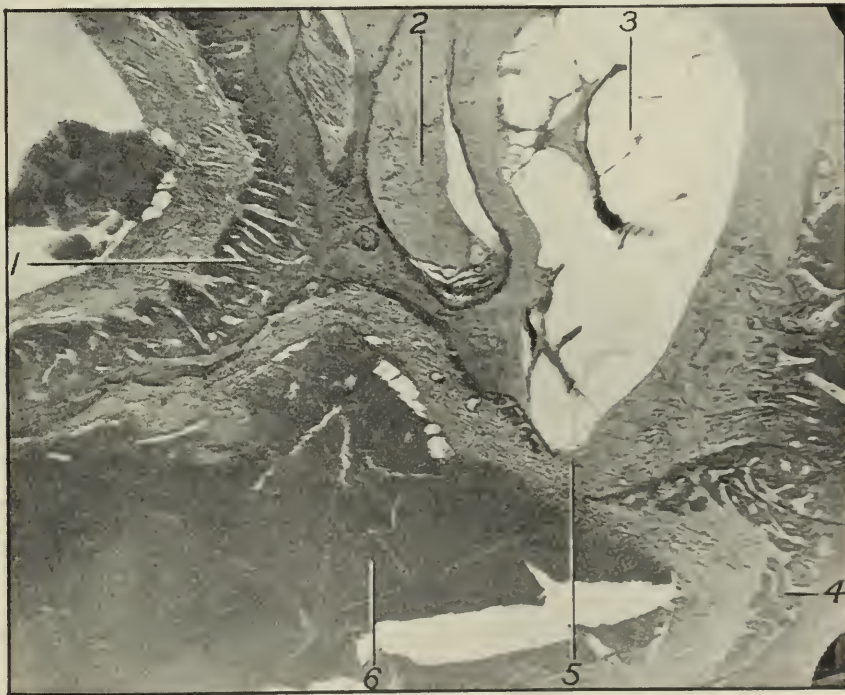


FIG. 16. — *Guinea-pig N*: Tubercular disease of the ear (experimental) (section 35). 1, new bone formation in external meatus; 2, facial nerve; 3, vestibule; 4, new bone formation in tympanic cavity; 5, early stage of fistula into external canal from middle ear—the bone has been completely eroded by tubercular disease, but the perilymphatic space is still normal; 6, pus in tympanic cavity.



FIG. 17. — *Guinea-pig N*: Tubercular disease of the ear (experimental) (section 45). 1, posterior part of footplate of stapes; 2, facial nerve; 3, anterior part of footplate; 4, basal coil of cochlea; 5, perforation of footplate of stapes and invasion of perilymphatic space of vestibule by tubercular granulation tissue and pus; 6, saccule; 7, utricle.

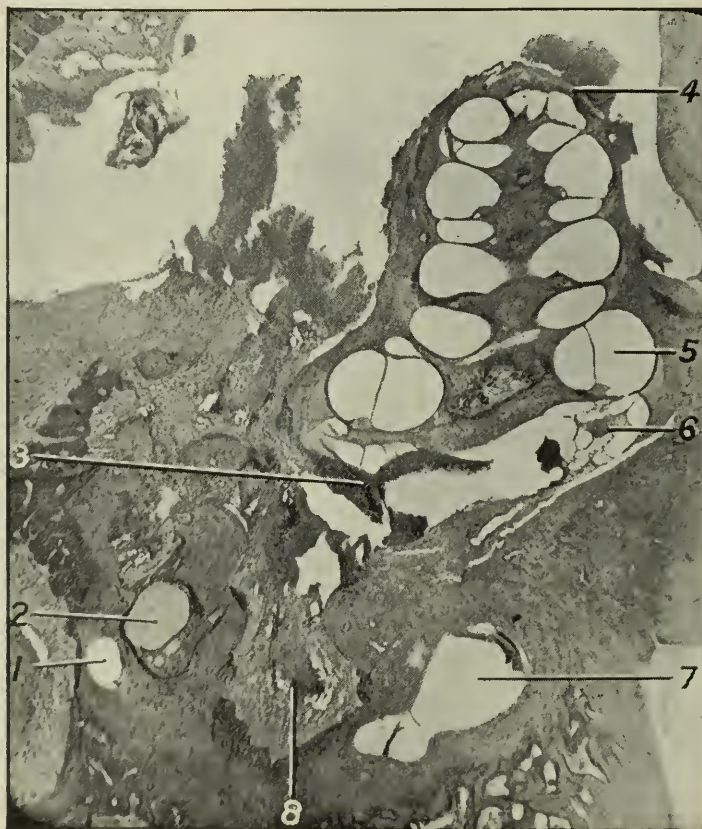


FIG. 18. — *Guinea-pig N*: Tubercular disease of the ear (experimental) (section 110). Horizontal section through right ear showing invasion of labyrinth through round window. 1, external canal; 2, facial nerve; 3, invasion through membrane of round window; 4, erosion of apex of cochlea; 5, dilated cochlear duct of basal coil; 6, new connective tissue in scala tympani of basal coil; 7, ampullary end of posterior canal; 8, sinus tympani with granulation tissue and pus.

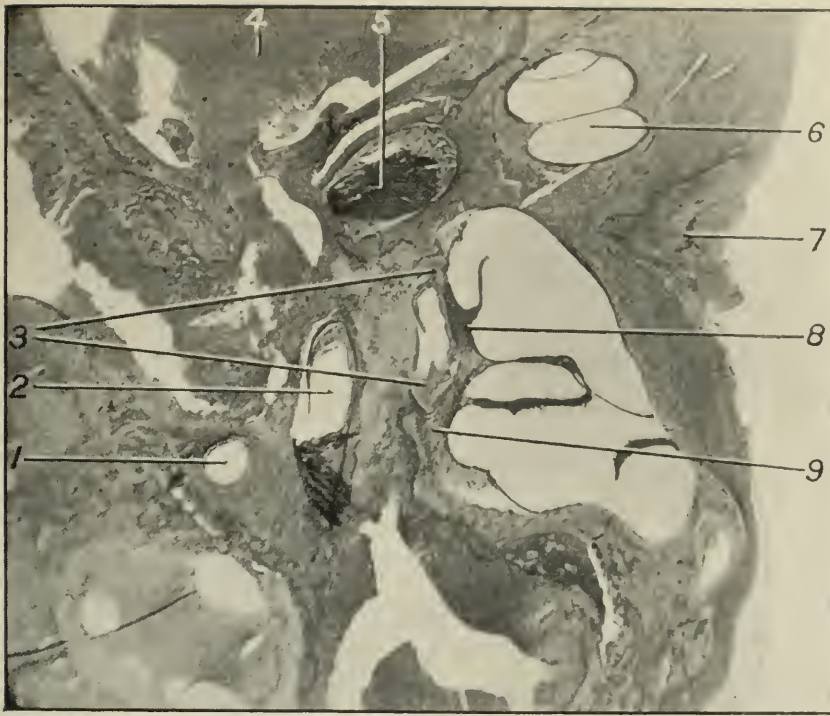


FIG. 19.—*Guinea-pig D*: Tubercular disease of the ear (experimental) (section 110). Horizontal section through middle and inner ear, showing invasion of vestibule by tubercular disease through posterior margin of oval window. Granulation tissue in vestibule. 1, external canal; 2, facial canal, the nerve is displaced (artefact); 3, footplate of stapes; 4, pus; 5, tensor tympani muscle; 6, basal coil of cochlea; 7, internal meatus, pachymeningitis; 8, tubercular tissue on inner side of stapes footplate; 9, tubercular tissue in perilymph space of vestibule.



FIG. 20.—*Guinea-pig D*: Tubercular disease of the ear (experimental) (section 155). Horizontal section through right ear, showing erosion of apex and outer bony wall of cochlea; infiltration of round window. 1, stapedius; 2, infiltration of round window membrane; 3, pus in tympanic cavity (bulla); 4, erosion of outer bony wall of cochlea; 5, new connective tissue in scala tympani of basal cochlear coil; 6 and 7, ampullary end of posterior canal opening into vestibule.

Guinea-pig P.—Right tympanic cavity inoculated through drum-head with tubercle bacilli emulsion on April 2, 1911. The animal died on May 22, 1911. Post mortem: Extensive caseation of lymphatic glands. The mucosa of the right tympanic cavity was greatly swollen. A few tubercle bacilli were found in the pus in the right middle ear. Sections 85 and 105 shown to illustrate invasion of labyrinth through oval and round windows.

Guinea-pig N.—The right tympanic cavity was inoculated with tubercle bacilli and staphylococci on February 11, 1911. The animal died on April 7, 1911, and the post-mortem examination showed well-marked tuberculosis of lymphatic glands, liver, spleen, lungs, and heart. Sections 35, 45 and 110 shown to illustrate invasion of labyrinth in region of external canal, oval and round windows.

Guinea-pig D.—Right tympanic cavity inoculated through drum-head with emulsion of human tubercle bacilli on November 17, 1910. The animal died on February 10, 1911. Post mortem: Tuberculosis of lymphatic glands, liver, and spleen. An extradural abscess was found in the middle fossa on the right side. The right middle ear was full of pus, which contained tubercle bacilli, streptococci, and staphylococci. Sections 110 and 155 shown to illustrate invasion of labyrinth through oval and round windows.

[The writer (J. S. F.) wishes to acknowledge a grant from the Carnegie Trust towards the expenses of this investigation.]

Specimens of Tuberculosis of the Temporal Bone.

By ARTHUR CHEATLE, F.R.C.S.

(1) RIGHT temporal bone of an infant who died of general tuberculosis: Tuberculosis of the lining membrane of the middle-ear tract. Through a perforation in the posterior segment of the membrane the lining membrane is seen to be thick and nodular. The middle-ear tract, including the anterior, contained cheesy pus. The vessels are intact. Sections of the lining membrane, stripped off the external semicircular canal, show tuberculosis. See next specimen. (89.1, Royal College of Surgeons Museum.)

(2) Drawing of a microscopical section of the lining membrane from

the preceding section, showing tuberculosis. (89.11, Royal College of Surgeons Museum.)

(3) Left temporal bone of an infant, who died of general tuberculosis: The middle-ear tract wall full of brown pus. Complete loss of the membrane and of the neck, short process and handle of the malleus and articular process of the incus. The stapes is in position. There is caries of the promontory over the round window. The interior of labyrinth was not invaded. (90.1, Royal College of Surgeons Museum.)

(4) Right temporal bone of an infant who died of general tuberculosis: An operation has been performed. The external canal and Fallopian canal are opened by the disease. The promontory is rough and carious and the round window is irregularly enlarged. The stapes has been lost. (91.1, Royal College of Surgeons Museum.)

(5) Left bone of an infant who died of general tuberculosis. An operation has been performed. The external canal is carious. The promontory is carious and perforated. The whole labyrinth was invaded, and secondary perforations have occurred through the superior semicircular canal to the middle fossa, and through the posterior semicircular canal to the posterior fossa. (92.1, Royal College of Surgeons Museum.)

Cases illustrating Tuberculosis of the Ear, shown after Operation.

By W. STUART-LOW, F.R.C.S.

Case I.—A child, aged 1. Operated upon on November 30, for septic mastoiditis. Family history of tuberculosis on mother's side. The child had profuse discharge since August, and there was marked absence of pain. The points of the operation were an extensive cortical mastoid operation previous to which the discharges were very effectively removed from the meatus, by means of suction by the exhibitor's vacuum suction pump, which apparatus was also used at the time of the operation, to suck up blood and purulent discharge in the interstices of the bone. The whole of the excavated surface was then freely swabbed out with a solution of chloride of zinc (40 gr. to the ounce), and the wound was left open and only lightly packed with sterile gauze. In the after-treatment the suction method was also employed both in the meatus and in the cavity of the wound.

Case II.—A child, aged 18 months. Operation four months ago for profuse discharge in the ear and also tenderness and swelling over the mastoid. Tubercle bacilli were found in the discharge, and extensive caries of the bone discovered on operating. A very thorough cortical mastoid operation was performed, preceded by repeated suction of the meatus by means of the same apparatus as mentioned in the first case. A solution of chloride of zinc was applied also in this case, and in the after-treatment the discharges were got rid of by the employment of the suction apparatus each time the case was dressed.

Case of Tuberculosis of the Auditory Apparatus treated by Permanent Drainage of the Lateral Ventricle.

By C. E. WEST, F.R.C.S.

L. G., FEMALE, aged $2\frac{1}{2}$, was admitted to St. Bartholomew's Hospital on May 23, 1914, with a history of pain and discharge occurring from the left ear on May 12, and left facial paralysis on May 19. There was complete left facial paralysis. On the right side a radical mastoid operation had been carried out at Guy's Hospital in February, 1914. There was a continued discharge from this ear, and the lymphatic glands in both anterior triangles of the neck were enlarged. A few days later an extensive radical mastoid operation was performed on the left side. The tympanic part of the facial nerve was found to be completely lost in a mass of soft granulations, and the whole of the region of the labyrinth was crumbling, and the jugular bulb was exposed. The child was very collapsed at the end of the operation.

Early in July a facio-hypoglossal anastomosis was carried out on the left side. This had had no effect on the paralysis. About July 25 there was a sudden complete right hemiplegia, with the general signs of a chronic meningitis, squint, retraction of the head, and unconsciousness. These features gradually passed away, but the hemiplegia continued with only slow improvement. The right facial paralysis continued complete.

On September 30 the left mastoid cavity was curetted out and a small sequestrum was found and was removed. The right cavity was also curetted.

On October 19 the voice became feeble and husky, and on October 20 the child had a prolonged attack of crying followed by

convulsions, mostly right-sided, and unconsciousness. The convulsions subsided only after administration of chloroform, and recurred in a milder form later. Next day the child was much better. A large temporal flap was turned down and the whole of the squama was removed on the left side. When the dura mater was turned down the leptomeninges were found to be intensely œdematous and a large quantity of cerebrospinal fluid ran away. The brain bulged strongly through the opening. In the posterior part of the exposed area the cortex appeared of normal colour, but anteriorly, over what would represent the lower part of the motor area, the colour was blue, and there was clearly some kind of cyst. This was punctured and was found to be an enormously dilated lateral ventricle. The fluid was allowed to run away slowly, the dura mater replaced but not sutured, and the scalp wound closed. On the next day the child was sitting up, taking food, and quite lively. The wound healed absolutely well, in spite of much tension and bulging. The bulging continued, and gradually became more tense.

On November 3 there was a recurrence of the fits. On November 11 the child was once more anæsthetised, and the scalp bulge was pierced by a long needle armed with No. 3 twist silk, the needle being passed right across the bulge and brought out through the skin some 2 in. beyond the edge of the old incision. Both ends of the silk were buried. One such line passed from above and behind downwards and forwards emerging in the parotid region; two others led upwards into the parietal region. There was an immediate and remarkable passage of fluid along the threads, producing an obvious œdema. The child again had no disturbance, but the amount of fluid passed continued so large that on November 18 the left eye was still almost closed by swelling. This has gradually subsided. Since that date the child's general health had been excellent.

Bacteriology: The material from the original operation was kindly worked out by Dr. Eastwood. Typical tuberculous lesions were produced in guinea-pigs, and inoculation experiments on rabbits showed that the type of tubercle bacillus was "human." Two other cases of tuberculosis of the petrous similarly worked out have proved to be of the bovine type.

An Example of Tuberculosis of the Auditory Apparatus in Children.

By E. D. DAVIS, F.R.C.S.

A LITTLE girl, aged $1\frac{3}{4}$, was brought to Charing Cross Hospital with right otorrhœa and caseating posterior, auricular, and deep cervical glands on the same side. A radical mastoid operation was performed twelve months ago, and at the same time the infected glands were removed. The glands proved to be tuberculous. The child made an uninterrupted recovery, and all the disease has been arrested. The operation area rapidly healed and has remained sound ever since.

A Post-mortem Specimen of a Radical Mastoid Operation per- formed Six Months before Death to illustrate Secondary Auditory Tuberculosis in an Adult.

By E. D. DAVIS, F.R.C.S.

THE patient was a comedian who had, for some years, suffered from pulmonary tuberculosis. During sanatorium treatment he developed mastoiditis and facial paralysis, following chronic otorrhœa. At the time of the radical mastoid operation he was suffering from advanced laryngeal and pulmonary tuberculosis. The mastoid process was extensively involved, and in removing the focus of disease a large area of the dura mater of the middle fossa was exposed. The post-aural wound healed by first intention, and the patient left the hospital after ten days with the symptoms relieved and health improved. When seen about four months before death the mastoid cavity was satisfactory.

The post-mortem showed extensive laryngeal, pulmonary, and intestinal tuberculosis. The middle fossa dura mater was thickened, and the exposed area covered by tuberculous granulation tissue. The petrous bone below the dura and surrounding the opening made at the operation was necrosed. The brain was normal and the meninges, apart from those in immediate relation to the area of operation, were unaffected.

A histological specimen of the granulation tissue stained for tubercle bacilli is shown.

DISCUSSION ON TUBERCULOSIS OF THE AUDITORY APPARATUS.

THE PRESIDENT (Dr. Albert A. Gray) said he always felt in some difficulty when the diagnosis of tuberculosis of the ear was in question. He admitted he occasionally judged a case by the result; if it got better he concluded that was, on the whole, against the idea of tuberculosis. He therefore hoped to learn much from this debate.

Dr. WYATT WINGRAVE said that the pathologist's share in the diagnosis of tuberculosis of the ear was mainly concerned in the examination of discharge and of curettage material. In the discharge one naturally looked for tubercle bacilli. This was often disappointing as compared with sputum, for they were rarely seen except in acute cases. In chronic cases, however, the discharge frequently presented acid-fast bacilli which had a striking resemblance to tubercle bacilli, and unless carefully stained and critically examined might lead to mistakes. They differed from tubercle in readily yielding the fuchsin to alcohol after differentiating in H_2SO_4 . In shape they varied considerably; some bore a striking resemblance to tubercle, while others might be thick or thin, long or short, massed, felted, fasciculated, or discrete. Further, they grew readily on agar, but lost their acid-fast property. They were not "colour-true"—i.e., they were apt to take up the counter-stain. In aural disease associated with atrophic rhinitis they must be specially looked for, as they were the most prominent and persistent organisms in this affection. In curettage material perhaps more reliable evidence was found, since giant cells or bacilli were easily seen, whereas in discharges giant cells were uncommon. One must be careful to distinguish between the genuine giant cell and the large multinucleated (syncytial) cell so often present in non-specific granulomata involving the bone (peri- and end-osteal). Giant cells were very common in chronic tuberculosis but rare in acute, exactly the reverse of tubercle bacilli, which were extremely rare in chronic cases. So much so that it was a working rule that in *chronic* tuberculosis giant cells were +, bacilli —; *acute* tuberculosis, giant cells were —, bacilli +. Instances of primary tuberculosis seemed specially to select the meatus,

probably the result of direct digital infection, since several specimens of curettage from meatal granulomata contained tubercle bacilli. It was rare to find the normal character in the discharge; instead of being "watery and curdy" it was far more frequently "creamy" or purulent, owing to the superadded "pyogens."

To ensure accuracy in the differential diagnosis of bacilli it was safer to use the micro-fuchsin method in preference to the Ziehl-Neelsen, which, unless very thoroughly and scrupulously followed, was unreliable. He now used the micro-fuchsin as a routine stain, it being easier to employ and facilitating search. The ear was not the only organ which sometimes failed to afford a positive diagnosis of tubercle, of which there might be very little doubt clinically, and in suspected primary cases von Pirquet's or other tuberculin tests were specially called for when bacilli were not found in either the discharge or the curettage. In chronic tuberculosis of the ear tubercle bacilli were found in 16 per cent. of the cases; in acute, 87 per cent.

[*Note.*—Simplified micro-fuchsin method: Substitute saturated alcoholic solution of picric acid for water solution of methylene blue. Wash freely before staining by fuchsin and after the acid bath.]

Mr. ARTHUR CHEATLE said that the series of specimens he showed demonstrated the spread of tuberculous disease through the temporal bone in infants. The first showed tuberculous disease of the lining membrane; the second, implication of the ossicles and promontory; the third, perforation of the outer labyrinthine wall and facial canal, and exterior to the dura mater of the middle fossa; the other showed implication of the labyrinth, while the last showed re-perforation of the labyrinth through the superior and posterior semicircular canal to the dura mater of the middle and posterior fossæ. He thought the cases of tuberculosis of the temporal bone in infants were generally bovine in origin and related a typical case which had been investigated by Dr. Eastwood, who reported: "The discharge on the dressing was extracted with saline and inoculated into a guinea-pig, which subsequently developed tuberculosis. From the guinea-pig a culture was obtained which, on being tested upon differential glycerine-bed media, exhibited the scanty growth typical of the bovine bacillus. Inoculated into a rabbit, the culture produced fatal generalised tuberculosis, which is also typical of the bovine bacillus." He believed the infection was due to milk, and occurred through the Eustachian tube. He said tuberculosis of the temporal bone in adults with lung tuberculosis ran

an entirely different course. This might be due to two reasons: a different organism or different anatomical condition of the bone, for there were very few adult bones in which the diploë could be attacked in the way which was so typical of the disease in infants' bones, in which the diploë was so open to attack. In adult tuberculosis with lung affection he had seen healing of the middle-ear infection under antiseptic treatment, but with very dense adhesions and greatly diminished hearing.

Mr. C. E. WEST said he only brought forward the case he had shown because he was asked to contribute something to the meeting, and it was something of a surgical curiosity. The method adopted to permanently drain the lateral ventricle had been a complete success. Mr. Cheatle's remarks had stimulated him to refer to one or two points. He, like others, had seen undoubted cases of tubercular infection of the ear in adults get well; but he had very rarely seen tuberculosis of the middle ear, in adults or in children, get well after they had suffered extensive loss of membrane and serious secondary infection. He agreed with Mr. Cheatle that the typical behaviour in an adult differed from that in the child; and one asked oneself why that should be. He had been inclined to imagine that Mr. Cheatle's explanation was a good one. He also had been having his cases followed up by Dr. Eastwood, and of the three cases he had had recently of proven tuberculosis—and only proven cases should have any weight—in children, two were typically bovine, the other, which he showed on this occasion, with tuberculous disease of the petrous bone and extensive destruction and invasion of the labyrinth, was human tuberculosis. Therefore, he did not think one could ascribe very much weight to the type of the bacillary infection in determining the clinical type; it seemed, rather, a question of soil and seed; a matter of the degree of resistance of the individual. In children, as it usually ran an acute course in them, there was destruction of the membrana tympani, and the difficulty of the exclusion of secondary infection was enormously augmented in the case of children, compared with adults, because children put their fingers into their ears after the fingers had been in all kinds of places and orifices of the body, so that there was usually a very mixed infection. On comparing cases of tuberculosis of the petrous bone with those of tuberculosis of the hip-joint, it would be seen there was a tremendous difference in the ordinary prospects of recovery. He believed that the essential point was the presence of secondary infection. The majority of the cases in adults which he had seen recover had had no perforation:

he had never seen a chronically open case of tuberculosis of the ear in an adult recover. He did not think there was any surgical progress in recent years in reference to tuberculosis of the petrous bone. If the child's resistance was good, it might recover, but not otherwise. The line which he believed the future would show was some more effective way of dealing with the bacteriological side of the question : the increasing of the immunity reaction.

Mr. E. D. DAVIS said the little girl he showed was a typical case of tuberculosis of the mastoid, with enlarged glands in the neck. It was one of the five cases whom Major Waggett and he had had at Charing Cross Hospital. The child had done well owing to the care which the mother had taken ; and in the other cases of the kind he had the patients also fortunately had good mothers. The specimen he showed was obtained from a patient with very severe pulmonary tuberculosis. A mastoid operation was done six months before death ; and there had been chronic otorrhœa and facial paralysis. About ten weeks after the mastoid operation the wound was more or less dry, and the patient went to the north of France, where sanatorium treatment was carried out ; but death ensued from pulmonary and intestinal tuberculosis. At the post-mortem examination it was found that the wound had not completely healed, there was some discharge, and granulations were growing from the dura mater which formed the roof of the mastoid cavity.

Mr. STUART-LOW said he had brought forward cases to illustrate what he considered to be successful surgery in tuberculosis of the ear, also because the cases illustrated some methods which he had found useful. There could be no doubt the cases were tuberculous. Agreeing that the route of infection in these cases was by way of the Eustachian tube, if the case was not too acute, and there was no mastoiditis, he thought the throat should be operated upon first. In this way the enlarged and septic tonsils and the often more septic adenoids were got rid of and re-infection of the aural cavity prevented. He was certain that many cases in which the ear again began discharging and ultimately became as bad as before operation were due to the tonsils and adenoids being left. If there were an acute mastoiditis, of course, this had first to be operated upon, but in such instances the enlarged tonsils and adenoids should be removed as soon as possible after the mastoid had been operated upon. The septic and often very fœtid discharge in the

meatus and tympanic cavity should be thoroughly got rid of before operation, and for this purpose he had long since given up syringing the meatus. There were dangers in syringing, as it was likely to drive the sepsis farther afield into the antrum. He now exclusively employed suction to remove these discharges—the suction was most effective, and he used it before operation so as to get a clearer field to work upon; suction was also most helpful during the operation to keep the wound free of blood and to get rid of the pus lurking in the interstices of the bone. The wound and whole bony cavity were then rubbed over with a solution of chloride of zinc, and left open, only a very light gauze dressing being applied and changed daily. No syringing was used during the after-treatment, only suction. In this way the discharge was thoroughly drawn off—no tension ever occurred, and the parts healed up very rapidly.

Dr. FRASER said that he had himself operated upon fifteen cases, with only one, or at most two, cures. He did a very radical mastoid operation, exposing the dura mater of the middle and posterior cranial fossæ as widely as possible, for the dura was the only tissue which formed a good barrier to the spread of tubercle. He treated the cases afterwards by administering sodium iodide to the child in its bottle, and packing the cavity with gauze soaked with peroxide of hydrogen, on the same lines as in tubercle of the nasal mucous membrane (Pfannenstiel).

Sir STCLAIR THOMSON said he did not feel that he could contribute anything to the debate, but for some time he had felt that he might have been neglecting the opportunities for studying the condition afforded by the King Edward VII Sanatorium. And yet he might not have been neglecting those opportunities, seeing that the disease was so very rare in the ear! When he began his work there he did not make a search for it, and more patients might have had the disease in the ear than he thought, because they did not complain of it. In three years he had seen there 800 patients, who had been admitted for tuberculosis of the lung. By excluding those who did not show the tubercle bacillus, the number might be stated as 700; 178, or 25 per cent., had tuberculosis of the larynx; but among the 700 patients he came across only two with what he took to be tuberculosis of the ear; the cases were painless, and the discharge only slight. The description of "watery and curdy" discharge

mentioned by Dr. Wingrave was new to him (the speaker), but it was a correct description of the discharge in those two cases, and of other cases which he had seen in private. Because of the painlessness and scanty discharge, there might have been other cases which he did not know of; though he did not think there could have been many, because all complaints were there carefully looked into. He hoped he might gather from the debate how, without the aid of animal experiment, one could recognise tuberculosis of the ear. The few cases he had encountered seemed to have been absolutely uninfluenced by sanatorium treatment, though he had seen only advanced cases, in which the prognosis was black from the beginning, and so treatment applied to the ear seemed useless.

Professor URBAN PRITCHARD said he knew but little of the new pathology and bacteriology, but he felt that, excellent as it was, it had one danger—namely, that it was apt to affect the diagnosis from the clinical standpoint. He had seen cases which, to an old stager, were undoubtedly tubercular, cases in which there was tubercle in other bones of the body, and which younger men doubted, from laying too much stress on the finding of the bacillus. He would therefore insist on the continuance of the study of these cases from the clinical point of view. Another point concerned the after-treatment in cases in which operation on infants was performed for tubercular disease of the mastoid. He agreed as to the immense importance of good general treatment, and he could illustrate that by a case. A very thin and wasted baby was brought to him, evidently the victim of tubercular disease of the mastoid; the wasting was so severe that it was of no use to attempt operation. But he prevailed upon the father to allow the child to be taken into the hospital under the care of Dr. Still, for if it could be got into better general health an operation might be possible. That was agreed to, and six weeks afterwards the child was so much better that he (the speaker) operated. A year later the child was again brought, simply a bonnie little boy. It had been well treated in the open air in the country, and had made a good recovery.

Dr. DUNDAS GRANT said he had been asking himself why, with the material at Brompton Hospital, he had not had more extensive experience of tuberculosis of the middle ear. In the adult it was a comparatively rare complication of pulmonary tuberculosis. He

thought the mode of infection in tuberculosis of the mastoid in children was chiefly through the blood; but with the shorter Eustachian tube in the child, and its increased patency, there was no reason why infection should not be by way of that tube, though the bone localisation seemed to suggest the blood as the medium. In the adult he thought it was chiefly through the Eustachian tube, and perhaps during coughing there was projection of material through that tube. This should not take place in the healthy person, because it was in a contrary direction to the action of the cilia and the tube was very narrow. In wasting diseases, however, the tube became abnormally patent owing to the absorption of the fatty layer in the wall and the action of the cilia might be disturbed. This question of route was not of mere academic interest, but was of great importance, both with regard to prophylaxis and treatment. He thought that in the case of the child the main factor in prophylaxis was looking to the milk supply (bovine tuberculosis), and the maintenance of the child's powers of resistance by every possible means. In the adult, the hygiene of the nasopharynx he regarded as very important, especially if the Eustachian tube was accepted as the channel of infection.

With regard to diagnosis, the occurrence of the discharge without pain, as mentioned by Dr. Logan Turner, was very important. The fallacy was that the discharge might occur without pain in cases in which, at a time previous to that which a patient could remember, there had been perforation of the tympanic membrane. Dr. Grant had found that often the disease was ushered in by deafness, and hardly any other sign. The tympanic membrane might then, perhaps present a grey, fluffy appearance, and on puncturing, typical caseous material would be exuded. Sometimes multiple perforations were seen, and in one otherwise non-characteristic case he got on to the scent of the tuberculous factor by the fact of there being two perforations.

With regard to the presence of bacilli, this need not indicate that there was disease of the petrous bone; and the absence of bacilli did not always exclude it. A small portion of tissue might be removed by means of Hartmann's miniature punch forceps for microscopical examination; he had shown such a specimen which the pathologist said was definite enough, and the after-history proved it. Bearing in mind the considerable proportion of cases in which suppurative otitis in tuberculous subjects got well, he thought there must be a number in which middle-ear suppuration must have been a coincidence, rather than part of a tuberculous process, and naturally the weak state

of the patient favoured the persistence of the discharge. Everything seemed to depend upon adopting hygienic treatment, and adopting a policy which was a complex of conservatism and enterprise. He thought pyoktanin had a beneficial effect in tuberculosis of the middle ear, and sometimes it was well to combine tuberculin with other treatment, but he would not use tuberculin for diagnosis in these cases. There was nothing worse than setting up a focal reaction in a bone which was so close to the meninges. As Professor Pritchard said, one avoided operating when the strength of the patient was at the lowest ebb; but a practical rule was laid down by Politzer with regard to the occurrence of tuberculosis of the middle ear in relation to pulmonary tubercle. If the ear trouble developed secondarily to the pulmonary, one should as a rule abstain from operation; but if the onset of the middle-ear disease was the first event, operation was advisable.

On the whole, he thought the kind of cases under discussion were to be looked upon with some hope; one should not regard every case of suppuration in the middle ear associated with pulmonary tuberculosis as necessarily beyond treatment. Many, he felt sure, were not tuberculous, and were susceptible to benefit from treatment, as also many of those which were actually tuberculous.

Dr. JOBSON HORNE reminded members that the subject was fully discussed by the Otological Society of the United Kingdom twelve years ago (February 2, 1903). The report¹ of that debate, to-day, might be read with advantage, and when read side by side with the report of the present debate it would be found that not much had been added to the sum total of their knowledge of the disease. That was not a disparaging remark, on the contrary, it was a fact which helped to prove that in primary tuberculosis of the ear—and that was the phase of the disease with which the debate had been mainly concerned—they were dealing with a disease which presented definite clinical symptoms and appearances, which occurred at a particular period of life, and which caused pathognomonic changes in the temporal bone. He had so recently² discussed in the Section the clinical and pathological aspects of the disease that he intended to direct his remarks more towards the ætiology of primary tuberculosis of the temporal bone.

¹ *Trans. Otol. Soc. of the U.K.*, 1902-03, iv, pp. 30-87.

² *Proc. Roy. Soc. Med.*, 1914, vii (Otol. Sect.), pp. 49 *et seq.* : *ibid.*, p. 62.

When opening the discussion twelve years ago he pointed out that the stress of the disease was more upon the mastoid bone itself, and suggested that the disease should be grouped under tuberculous diseases of bone. In the same discussion Mr. Hugh Jones referred to a paper by Dr. Nathan Raw in the then current number of the *British Medical Journal*, pointing out that bones and joints were more susceptible to bovine tuberculosis. Since then further evidence had been brought forward in support of that view, and that evening it had been stated that bacteriologists regarded primary tuberculosis of the ear as more probably due to bovine than to human tuberculosis. In addition to the findings of the bacteriologist, Dr. Horne went on to say, there was clinical and circumstantial evidence of a most valuable nature in support of the bovine origin of the disease. In the first place, the age of incidence of primary tuberculous disease of the ear. If an average were struck of the ages at which those cases came under notice it would, in his opinion, be found to be between one year and fifteen months, that was to say, the age of incidence of the disease would be during the period of the milk diet of life. Upon further inquiry it would be found that the majority of the cases occurred in bottle-fed and not in breast-fed children. That latter fact had been brought out by the valuable statistics supplied by Dr. Logan Turner and Dr. Fraser from Edinburgh.

A piece of circumstantial evidence in support of the bovine nature of the disease was, that cases of primary tuberculosis of the ear so seldom came under notice in private practice, that was to say, from amongst a class that carefully considered the milk supply. The solitary exceptions that occurred, when investigated, supported the bovine theory. Another piece of circumstantial evidence that supported the bovine theory was the enormous discrepancy in the frequency with which primary tuberculosis of the ear was met with in hospital clinics in different districts. Upon inquiry, those discrepancies were fully explained by the amount of slum and impoverished districts which the hospitals immediately served. Lastly, the enormous involvement of the periotic lymphatic glands supported a bovine origin of the disease.

There were other points with which he would have dealt had time permitted, but he thought he had said enough to show that even if the debate had not added to the sum total of their knowledge of the subject which they had been discussing it had, at least, been the means of co-ordinating facts in support of the views which they had held.

Mr. RICHARD LAKE said that in his last case of tuberculosis in a child, he had added tuberculin to its diet, and the result had been very good, though he could not say what part the tuberculin played. Sir StClair Thomson and Dr. Horne had both spoken on a point it had been his intention to raise—namely, the great rarity with which one saw tubercular disease of the ear among patients in a sanatorium. When he ceased his connexion with the Mount Vernon Hospital he had seen 600 cases of laryngeal tuberculosis, but not one of tuberculous disease of the ear, although one or two patients had a discharge from the ear: though there might have been a bacillus or two in the discharge, he did not consider them tubercular. He believed the reason was that people had to wait a long time before they could get admitted to a sanatorium, and if their case was acute they died before admission. His private practice showed him the same thing—ear trouble was found more in acute cases. One might say that in adult aural tuberculosis the chance of recovery varied inversely with the acuteness of the chest trouble. A very interesting case was that of a man who had been shot through the lung in the South African War. He contracted tuberculosis of lung, larynx, and ear; there was a tuberculoma on the false cord. He was taken into hospital, and his mastoid operated upon. Dr. Peters removed the tuberculoma, and now, four years later, he was still well. When there was active lung trouble it was not wise to operate on the ears.

Mr. MARK HOVELL said that, in his experience, tuberculous disease of the ear was comparatively rare. The last case of the kind he saw was that of a child aged 9 months, which had been fed, almost entirely, on milk from a neighbouring farm. There was no other source of infection, and everything pointed to the milk as the source of mischief.

Mr. E. D. DAVIS, referring to Dr. Fraser's practice of using sodium iodide and hydrogen peroxide, said Major Waggett and he had been in the habit of painting the mastoid operation cavities with iodine, and packing with gauze soaked with hydrogen peroxide. Major Waggett and he thought the results were much better. At Mount Vernon Hospital he saw about 500 advanced cases of laryngeal tuberculosis, but not one of tuberculosis of the ear, though there were some cases of otorrhœa.

The PRESIDENT expressed the indebtedness of the Section to the gentlemen who had participated in the discussion. Several speakers had, unconsciously, supported one another in certain matters — for instance, the likelihood that the disease was bovine tuberculosis in infants and human in adults, and the infrequency of tuberculosis of the ear among sanatorium patients. Contaminated milk certainly seemed to play a large rôle in the production of this disease in children. He agreed with Dr. Grant as to the rarity of the disease in adults. For a year he went to a large Glasgow Poor Law hospital, where all the phthisis cases were segregated, for the purpose of seeing cases of non-suppurative ear disease, otosclerosis, and nerve affections of the ear, and he was struck with the relative rarity of suppurative disease in phthisical patients. The remarks as to the frequency with which bottle-fed infants were attacked bore out the idea as to milk being the common mode of infection. There was much to be said in favour of Dr. Pritchard's remark as to the importance of *clinical* diagnosis, since many of the latest laboratory tests were intricate and required some time for performance. The discussion had been to him most instructive and interesting.

Mr. STUART-LOW said that he believed cases could be cured by operation, as shown by the two cases he had exhibited that day, but there was the danger attendant upon the children returning to their unhealthy homes, and for this supervision was necessary. With regard to cow's milk, calves were not tuberculous when born, nor even as late as two years of age. This being so, a fresh breed of milk cows could be reared and so tuberculous milk would cease to be produced.

Dr. FRASER said that their cases were practically all in children under 2 years of age (nine adults to fifty-one children was their proportion), and to ascertain whether very young children were deaf was not an easy matter. Between the infantile bovine cases on the one hand, and those associated with advanced phthisis pulmonalis on the other hand, there were scarcely any cases of tubercle of the ear. His belief was that the treatment of early cases was preventive, as operation offered little chance of permanent cure. If one had to send children back after operation to their unhealthy homes, such as those in the slums of Edinburgh, one felt the hopelessness of operation. But if they could be sent to a sanatorium afterwards, it was worth while to operate. Ruttin, of Vienna, had told the speaker that if there was

pain in these tuberculous cases they operated ; if no pain, no operation was done, because a cure was not to be expected. One could not get all the granulations away, nor the last vestige of disease. In regard to the prevention of tuberculous ear disease in infants, 20 per cent. of the Edinburgh milk was tuberculous, and it was said it would cost the nation £40,000,000 to ensure a milk supply free from possible contamination by tubercle. Statistics compiled by John Fraser and Mitchell with regard to tuberculosis of bone and glands in children showed that 70 per cent. of tuberculous bone disease and 90 per cent. of tuberculous cervical gland disease in children was of bovine origin. If all tuberculous cows were slaughtered, following a tuberculin test, there would be little or no need for operation on tuberculous otitis media, as the cause would have been eliminated.

Otological Section.

May 14, 1915.

Dr. ALBERT A. GRAY, President of the Section, in the Chair.

A Note on Mastoid Grafting.

By DAN MCKENZIE, M.D.

THIS note merely deals with the mode of retaining the graft. I practise immediate grafting, as Mr. Ballance advises, and there is no doubt that it shortens convalescence. After the graft has been accurately apposed to the walls of the bony cavity, all that need be done to secure its retention is to allow the cavity to fill with blood. The coagulum retains the graft perfectly in position, and packing is therefore unnecessary. In cases where the cavity has had to be re-opened, a few days after the operation I have always found, on removing the coagulum, that the graft is comfortably *in situ*. Naturally, one must be careful to make sure that bleeding from the osseous surface *under* the graft is not taking place. In the subsequent dressings the blood-clot is left undisturbed. It begins to disintegrate four or five days after the operation, and when that process is completed the graft will be found to have sown its cells over the surface.

DISCUSSION.

Mr. STUART-LOW asked how the grafting was carried out. If the grafting were done at the time of operation by inverting the skin dissected up from the surroundings of the wound he did not approve of the method, as skin could never be rendered properly aseptic and would therefore endanger the wound. He had known of instances where erysipelas had occurred when this method had been followed. Since he had introduced the blood serum method in his

operations (filling the bony cavity with blood serum) he had got better results than by grafting, as the average time that the bony cavity was cicatrised over was one month, and the hearing three months after the operation was much better than after skin-grafting.

Mr. MOLLISON wished to support the practice of Dr. McKenzie of grafting immediately at the operation. He had watched Mr. Marriage apply the graft with the suction apparatus, and the apposition of the graft was perfect. Finding that the suction apparatus often broke, he (Mr. Mollison) now used gauze, which held the graft in place just as well. If he found that Dr. McKenzie's blood method held the graft well, he would cease his attempt to put in gauze, as it was a difficult procedure. Undoubtedly a skin-graft put into the cavity facilitated healing afterwards, particularly at the raw cut meatal edges; instead of getting a raw area to heal with granulations at the entrance of the ear, a perfectly healed skin margin was produced in three or four days.

Dr. DUNDAS GRANT asked whether Dr. McKenzie made use of adrenalin, in order to prevent bleeding at the time of applying the graft. And if so, did any reaction take place, as was seen in some other cases. With regard to the warning as to bleeding occurring under the graft, had his observation led him to regard that as important? He thought the pressure exercised by the clot on the graft could not be so complete as that exerted by the gauze plug. He (the speaker) had always resorted to a gauze plug, but he would be glad to dispense with it.

The PRESIDENT said he did not know when Mr. Ballance devised his method of immediate grafting, but Dr. Dench, of New York, told him he used to carry out the method seven or eight years ago. When he (Dr. Gray) was speaking at the International Congress at Budapest that writer warned him that sometimes the graft did not take, and then it caused trouble, and had to be removed. To his mind also had occurred the question why blood-clot should keep the graft in place better than did gauze packing. The recommendation that bleeding from the osseous surface under the graft should be arrested was a counsel of perfection. Could bleeding from the osseous surface be stopped except by clotting in the veins? Adrenalin did not seem to contract blood-vessels in bone.

Dr. H. J. DAVIS said it would be interesting to hear from members whether they were accustomed now to graft or not. He did not do it, though he did not object to it; it was merely that he did not see the object of it unless the bone cavity was so large as obviously to require it.

Dr. KELSON said his impression was that since Mr. Ballance brought out his classic work the practice of grafting had gradually declined; and he agreed with Dr. H. J. Davis that it would be most interesting to ascertain whether otologists as a body now grafted.

Dr. DAN MCKENZIE replied that he of course used the Thiersch graft, not the entire thickness of skin. If members who did not practise skin-grafting were to try it in a few cases, he thought they would become converted to it. It needed some manipulation, but with the suction apparatus it was wonderful how well it could be aspirated into position. Although, theoretically, the chances of sepsis occurring would be increased by putting a graft on a raw surface of bone before a protective layer of granulations had formed, he did not recall a case of his in which he could attribute subsequent sepsis to the graft. Before he began to graft, suppuration after mastoid operations was as common in his cases as it was now. One could very seldom get a mastoid cavity aseptic; it could not be made aseptic. Mr. West had pointed out that when there was a fistula in the external canal, it was well to keep that free of the graft; and the same held good when there was a pocket of disease; any spot likely to be troublesome should be left bare. He did not use adrenalin in these cases, because of the risk of subsequent reactionary hæmorrhage, which would cause the graft to be floated up. He had not seen one float up, but the possibility of it could not be denied. If the bone surface was fairly dry, there was no obvious bleeding, and the clot which formed on the outer side of the graft prevented any great hæmorrhage taking place beneath afterwards. What Dr. Davis said was true, but the reason the graft did not take sometimes was that the wound had been so septic at first that it had killed the graft. In some cases he had had to take the graft out, or it had been accidentally removed in the dressing; and in those cases it seemed as if the cells from the graft had already distributed themselves over the surface. So when the membranous graft was removed, there were left a large number of epidermal cells which quickly took root and grew. He could not say what was the average time required for the wound to heal; perhaps the shortest time was about four weeks. There might be occasion to re-open owing to some complication such as a lateral sinus trouble, for example.

Case of Congenital Syphilitic Deafness undergoing Thyroid Treatment.

By J. DUNDAS GRANT, M.D.

THE patient, a young woman, aged 19, became suddenly deaf when aged 9; there is no recollection of attacks of giddiness. Her eyes had been affected for twelve months previously. The central incisors are deeply notched and slightly pegged.

She was first seen by Dr. Grant on November 13, 1914. The loud conversational voice was heard at 2 in. on the right side, and the whispered voice not at all. On the left side there was no hearing whatever. The caloric test showed slightly diminished activity on the right

side and considerably diminished activity on the left (i.e., nystagmus produced by cold air on the right side in thirty-five instead of twenty-eight seconds, and on the left in forty-nine seconds).

She had had slight snuffles at birth.

The first child was a miscarriage; the second, a boy, aged 21, alive and well; the third, the present patient; the fourth, a boy, alive and well; the fifth, stillborn, premature; the sixth, boy, died, aged 7 months; the seventh, boy, alive and well; the eighth, girl, alive and well.

She was ordered pil. hydrarg. and opium night and morning, and half a 5-gr. thyroid "tabloid" night and morning.

At the end of a fortnight the whispered voice was heard at 4 in., and then she took the thyroid "tabloid" increased to one whole tablet night and morning. A week later the whispered voice was heard at 4 in. on the right side, and the conversational voice over 5 in. Galton's whistle is not heard below the mark 7. The tuning fork on the mastoid is -3 seconds, and Rinne's test is positive shortened. Tuning fork on the vertex not heard.

She did not come for treatment from December 18 until to-day.

She is brought forward to-day with a view to estimating the result when she is exhibited on a subsequent occasion.

Case of Hereditary Syphilitic Nerve Deafness undergoing Thyroid Treatment.

By J. DUNDAS GRANT, M.D.

MRS. H., aged 26, first seen on July 17, 1914, complaining of noise and deafness in left ear of three years' duration and giddiness and sickness of six months' duration. The whispered voice was heard at a distance of 20 ft. on the right side and 5 in. on the left side. Rinne's test was positive on the right side, and negative reversed on the left; bone-conduction was normal on the right side and diminished (-3 seconds) on the left. The tuning fork on the vertex was heard loudest in the good ear. Galton's whistle was heard at 1.6 on the right side and 2.2 on the left. There was no narrowing of the left Eustachian tube and no improvement after inflation. The caloric test (cold air) produced nystagmus after twenty seconds on each side (more marked on right). The pointing test was normal. The central incisors are seen to be pegged.

She was at first ordered pot. brom., then pot. iod. and pot. brom., but the giddiness still persisted; she then took $\frac{1}{2}$ gr. of quin. sulph. three times a day for a fortnight. The giddiness ceased, and the nystagmus following the caloric test to the left ear was only produced in thirty-four seconds. The noises still continued; they were found to be slightly diminished by vertebral pressure, and she was again ordered pot. brom.; but the giddiness and sickness returned. She then went back to the quin. sulph., with the result that the giddiness disappeared, but the noises were worse.

She was then ordered 15 minims of liq. hydrarg. perchlor. three times a day for a week, and at the end of that time the head felt clearer and the noises were less. She took this till November 20, when she was ordered thyroid extract "tabloids," 5 gr. (half a "tabloid" to be taken night and morning for a week, and a whole one night and morning for another week), also to continue taking the liq. hydrarg. perchlor. At the end of the fortnight the head felt much clearer and the noises were less.

On December 18 her hearing for the whisper was at a distance of 7 ft. on the left side. She continued with the same treatment till January 8, 1915, when the hearing for the whisper on the left side was at a distance of 16 ft.

On January 8 she was ordered to continue the thyroid "tabloids," and to take pot. iod. 3 gr., liq. hydrarg. perchlor. 30 minims, inf. quassia $\frac{1}{2}$ oz., three times a day for a week. At the end of this time the hearing was not quite so good (6 ft. for the whispered voice), and she complained of headache and weakness in the head. She then took simply infusion of quassia for a fortnight, but was not so well at the end of this time.

On January 29 she was ordered thyroid "tabloids" and liq. hydrarg. perchlor., 15 minims, but at the end of a fortnight the hearing was only at a distance of 6 in. for the whisper on the left side. She then took the "tabloids" alone for a week, when the hearing was found to be at a distance of 5 ft. for the whisper; after another fortnight of this same treatment it was 8 ft. She was then ordered mist. pot. brom. in addition to the "tabloids" for fourteen days, and the hearing improved to 16 ft. for the whisper, while the noises were less.

She has been continuing with this treatment up till the present time. The hearing when tested a week ago was at a distance of 10 ft. for the whisper.

Dr. Dundas Grant added that his object was to show the cases to

enable members to judge what the future effect might be. He did not know what was the action of the thyroid, but in Rendle Short's "Newer Physiology" there occurred this passage: "The explanation of the effect of iodides on gummata, arterio-sclerosis, and aneurysm, is really the increased internal secretion of the thyroid gland." The doses and frequency of administration varied with the tolerance of the individual. He had not given more than a 5-gr. "tabloid" three times a day. In one case its use was followed by considerable improvement; the girl also sprang up in stature, and her general condition and strength were altogether better.

DISCUSSION.

The PRESIDENT said he had not tried thyroid extract in syphilitic cases. One of Dr. Grant's cases was having mercury also. He could not help feeling that the explanation given of the action of the iodide was only partially true, and that its effect on the viscosity of the blood was in part the explanation of its curative value in syphilis. Physiologists often found things which physicians did not. Iodide did not exercise any therapeutic value in tissues the cells of which had no power of multiplication; whereas it would affect syphilitic conditions in which cell multiplication was retained. If the cells of a tissue could not multiply, he did not think anything would put them right when once the nuclei were destroyed. The method mentioned by Dr. Grant supplied a useful hint for dealing with cases of syphilis which were resistant to treatment.

Dr. DUNDAS GRANT replied that, as the President had said, iodide did not always act; and the reason for that might be that in the particular individual there was not enough of the thyroid gland to react to it. Where iodide did not act, the addition of thyroid extract from without supplied what the person was not producing himself.

Congenitally Deaf Boy improved under Treatment.

By RICHARD LAKE, F.R.C.S.

MR. LAKE said the case had been shown before,¹ and if any member wished to have all the particulars he would supply them, but it would mean a good deal of work.

¹ *Proceedings*, 1914, vii, p. 41.

Aberrant Chorda Tympani.

By RICHARD LAKE, F.R.C.S.

A LADY, aged 40, with bilateral abnormality of the chorda tympani. The chorda passes from close to the umbo backwards and downwards and appears like a white bar moving with the tympanic membrane on



Bilateral aberrant chorda tympani.

rarefaction and condensation of the air in the external meatus. The abnormality was without significance or effect. Mr. Kelly and the exhibitor have described this condition.

DISCUSSION.

The PRESIDENT said he did not know of any cases of the kind other than those referred to in the notes. He did not know why the chorda tympani should sometimes take the course which it did in these cases.

Mr. LAKE replied that in the previous case he could not find by dissection any trace of the tensor tympani.

Pyrexia after Mastoid Operation for Acute Otitis Media.

By N. TURNER and R. LAKE, F.R.C.S.

THE patient, a young gentleman, a native of San Salvador, of a highly nervous temperament, developed an acute follicular tonsillitis towards the end of April. At the end of about ten days a large snow-white patch appeared on the anterior aspect of the right tonsil, followed by pain in the right ear extending down the radius of the lower jaw on the same side and causing an intense pain. This was on March 6. This patch was diagnosed as a pneumococcus infection, and treated accordingly.

On March 7 the aural symptoms had increased in severity, and

the drum was bulging. It was freely incised under chloroform, giving exit to a free flow of blood-stained serum. The pain and discomfort were relieved, but on March 10 some tenderness was noticed in the mastoid of that side, followed rapidly by redness, tenderness, swelling, and œdema, which had markedly increased by March 11, and there was acute tenderness in the region of the mastoid process on the next day, together with an enlarged gland in the neck.

His temperature had begun to rise, and he was sent into a nursing home on March 12 and the mastoid opened that evening. At the time of the operation his temperature was 102° F. A very small quantity of pus was found in the mastoid, but the whole bone and mastoid process were engorged and the cells filled chiefly with a sero-sanious fluid. A very free operation was performed, and the patient's temperature had fallen by the middle of the next day to 99° F., rising at night to 101° F. He was immediately given calomel 5 gr., and quinine 10 gr., repeated thrice daily.

On March 14 the temperature had gone up to 103·6° F., when the wound was dressed and everything was found looking extremely satisfactory. After dressing, the temperature fell one degree, rising in the early afternoon to 104° F., falling again at night to 101° F. During the next morning the temperature again rose, to 103·2° F., falling a degree and a half in the afternoon and rising to 103° F. again at night. He was then given 1 gr. of opium and 5 gr. of calomel. The next day his temperature steadily fell, with a small rise towards the evening of March 16, the rest of the convalescence being uneventful, and he now can hear as well on that side as on the other.

The point of interest, of course, in this case is what was the cause of his rise of temperature? When seen on the evening of March 15 we were prepared to make a lumbar puncture, but on a careful examination we could see no reason for his not doing quite well, and so no examination was made of his cerebrospinal fluid. Both of us were quite convinced that we had to deal with a case of threatening meningitis.

With regard to these post-operative pyrexias, one often sees cases in which operation on the mastoid is followed by a series of high temperatures; these, as in this case, rising considerably over 104° F., but without any rigors. This rise is accompanied by a marked increase in the pulse-rate and frequency of respiration, though sometimes the pulse-rate is but slightly altered. In these cases one cannot help wondering what would have been the result of a lumbar puncture, and one would feel inclined to advocate and carry out the methodical thecal puncture

in all cases in which there was a high pyrexia following mastoid involvement. These cases are so frequent that it seems very doubtful as to what is the proper line to adopt; whether to perform a lumbar puncture, and, if one found the reaction of the fluid neutral and that at the same time it failed to reduce Fehling's solution, then, would one in all these cases do a decompression operation, or what would be one's position?

DISCUSSION.

Dr. DAN MCKENZIE said the question the case raised in his mind was as to whether there was not some lateral sinus thrombosis. He believed it was agreed that lateral sinus thrombosis could disappear spontaneously. He could recall the case of a young lady who had a cortical mastoid operation done. It healed up well. Three or four weeks after the operation she developed symptoms of lateral sinus thrombosis, a swinging temperature, and at least one rigor. She was kept under observation; the temperature came down to normal, and nothing else happened. Some time after that he read in a foreign journal of similar cases, in which lateral sinus sepsis spontaneously got well. He would like to hear what members thought of the explanation, and whether they had had any similar experiences.

Mr. E. D. DAVIS said that a nurse at Charing Cross Hospital had a mastoid operation done. On the second or third day she had a temperature of 103° F., and it continued three or four days. At the operation the lateral sinus was exposed and regarded as normal; but when the temperature had been running about five days the sinus was compressed by a gauze plug, and after that the temperature came down and remained normal. The plug was placed under the bone in the lowest part of the sinus groove. The sinus was never opened.

Mr. MOLLISON said he saw no reason for invoking lateral sinus infection in these cases; it seemed to be simply a matter of infection *versus* resistance. He had seen cases of high temperature with very little to account for it; in some cases one could find nothing wrong, except a little catarrh in the middle ear. Since Christmas last he had seen a case similar to that shown by Mr. Lake. A young soldier had acute otitis media on both sides, the whole illness having started with a very high temperature, alarming vomiting, and headache. Both membranes were ruptured, but still the temperature remained very high. He opened one mastoid, and found extensive suppuration. As the temperature still persisted he opened the other; in this, too, there was similar extensive suppuration; still the temperature did not drop. He exposed both lateral sinuses and found them healthy. Infection proved to be a pure streptococcus. The patient eventually responded to vaccines and the temperature dropped.

Dr. H. J. DAVIS said that rigors were the chief indication of sinus disease. As a matter of fact, it seemed very difficult to infect the lateral sinus. Even in cases where the sinus was bathed in pus and covered with granulations

thrombosis rarely occurred. This was, he supposed, due to the enormous blood-stream and the rapidity with which infection was carried away from the seat of the trouble.

Mr. WHALE said he recently had a case which seemed to have been of the same nature as those mentioned by Mr. Mollison and Mr. E. D. Davis. The mastoid had been, elsewhere, explored on account of disease, and on the tenth day, while the patient was convalescing, the temperature suddenly rose and remained up. On four successive days it was 104° F., on the fifth day 103° F., and the next day 105° F. He therefore exposed the sinus down to the bulb, but found nothing, and when the child was put back to bed the temperature fell as suddenly as it had risen. The patient had remained well since. A physician had failed to find anything wrong in the chest or abdomen.

Dr. DUNDAS GRANT asked whether there was any headache in this case: in his own cases of meningitis the headache was severe. The temperature in the cases mentioned seemed to have been more or less continuously high; there were not the extreme oscillations characteristic of infection of the lateral sinus. The note said: "On careful examination we could see no reason for his not doing quite well." With a skilled clinician like Mr. Lake that meant much, and yet it seemed to need some courage to abstain from opening, and possibly exploring, the lateral sinus in this case.

Mr. LAKE, in reply, said he regretted that the year 1914 was missed out. He wanted the views of the Section on these cases, and thanked the members for what they had said.

Note on the Case of a Man, aged 43, after Operation for Ménière's Symptoms.¹

By W. H. KELSON, M.D.

PATIENT was a painter, and had been totally unable to follow his occupation owing to giddiness of aural origin. The operation of uncapping the external semicircular canal on the right (deaf) side was performed, and when shown at the Section he was free from giddiness. During the discussion, the question as to the probable permanency of the benefit was raised. In order to clear up this point the operator had made inquiry, and the patient, who lives at a distance from town, writes: "It is now over a year since my operation and I am able to be back to my work. I have not seen my doctor for several months. I am also feeling very much better in myself, and my friends think I am a marvel."

¹ Shown at the February, 1914, meeting of the Section, see *Proceedings*, 1914, vii, p. 43.

DISCUSSION.

Mr. E. D. DAVIS asked whether this patient was neurotic, and whether Dr. Kelson considered that uncapping the semicircular canal had cured the case.

The PRESIDENT asked whether there was any lead-poisoning associated with the case, and whether the giddiness was of long duration.

Dr. DAN MCKENZIE asked whether the vestibular system had been tested since the operation. Some years ago he noticed when a mastoid operation was done in a case in which there was a labyrinth fistula, that frequently when tested five or six months after the operation the vestibular system was found to be altogether in abeyance. If that were so, then in this case it would account for the vertigo having passed away.

Dr. KELSON replied that the patient had not been able to follow his occupation as house painter for over two years. The man was very much better after the operation than he expected to find him. The doctor sent him up with the statement that he could not do anything for him, and that he could not earn his living. A member (he believed it was Mr. Jenkins) had done the same operation in a case with considerable success. He did not consider the man was shamming, as he seemed so anxious to work. He was now again at work.

Acute Purulent Meningitis ; Drainage of the Meninges ; Recovery.

By DAN MCKENZIE, M.D.

THE patient, a male, aged 50, was operated on for chronic suppuration in the left ear, on September, 1914. A mastoid operation was performed, the "bridge" being left, and the patient left hospital convalescing.

After attending as an out-patient for some weeks he was readmitted on October 7, 1914, with a re-infection of the partially healed left ear. At the same time the right ear became the seat of acute purulent disease, with pain and discharge.

After simple treatment had been tried, on October 22, his temperature being sub-febrile, the radical mastoid operation was completed in the left ear, and at the same séance the same operation was performed on the right ear. On this side there was pus under pressure in a deep

mastoid cell, and here an eroded bony sequestrum, 1 cm. in diameter, was found and removed.

Next day (October 23) the patient was complaining of headache and the temperature was running between 100° F. and 102° F.

October 24: Seen by Mr. Kisch in my absence. Spontaneous nystagmus to the left. Some of the stitches in both of the wounds were removed to relieve tension, as they were inflamed and angry-looking.

October 25: Vertigo and diplopia complained of. Patient rather talkative and "strange." Severe headache on the right side of the head radiating down to the neck. Very deaf, hears a shout only. Temperature rose to 103.8° F.

October 28: Spontaneous nystagmus II to the left. Some rigidity of the neck and tremor of the head on flexing the head on the chest. No diplopia; no dysdiadokokinesis; Babinski's sign negative. Hears the tuning fork by bone-conduction in either ear with the noise machine. Complains of severe headache, and is excitable. No paralyses, but some intentional tremor of limbs present. Lumbar puncture: 20 c.cm. of turbid fluid withdrawn.

Subsequent examination of the cerebrospinal fluid by Dr. Wyatt Wingrave: Specific gravity 1008, sugar absent, proteids present; leucocytes numerous; lymphocytes, a few; endothelium; bacteria, numerous—cocci and Gram-negative bacilli. Culture, streptococci.

Operation: The headache being right-sided was the only indication on the part of the peccant ear. The right mastoid was therefore re-opened, and the posterior wall of the mastoid process removed to expose the lateral sinus and the dura medial to it. The vessel was opened, inspected, and found to be healthy. Double vestibulotomy was performed and the modiolus broken through to reach the internal auditory meatus, into which a wire drain was inserted. A transverse incision was made extending from close to the internal auditory meatus to the lateral sinus in the dura of the posterior fossa, and from the internal end of this incision a free flow of cerebrospinal fluid welled up. The wound was packed with dry gauze and left open.

October 28: Right facial paralysis. Spontaneous nystagmus to the left still persists. Patient very restless, and complaining of severe headache.

October 30: Pulse markedly intermittent, and patient very deaf and stupid. He is still restless and complaining of severe occipital pain. The appetite is good. The wound is draining cerebrospinal fluid very freely, the dressings being always soaked.

Temperature showed a gradual decline from 103° F. on October 28 to 99° to 100° F. on November 1. Thereafter for a couple of weeks it ran about 99° F., and on November 18 fell to normal for good.

November 1: Mentally clearer. Still headache, but less severe. Cerebrospinal fluid still draining.

November 4: Wire drain removed from internal auditory meatus.

In this case the translabyrinthine flow of cerebrospinal fluid was disappointingly slight. From the dural incision, on the other hand, the drainage was very free, continuing in gradually lessening quantities for about ten days. The symptoms declined in severity *pari passu* with the fall in the temperature. He is unable to remember anything of his illness during the week subsequent to the operation.

DISCUSSION.

Mr. WHALE referred to the last paragraph of the notes: "the translabyrinthine flow of cerebrospinal fluid was disappointingly slight," and asked whether the exhibitor did not consider that the cure in his case was due simply to cutting the dura, and that inserting a wire drain in the internal meatus did not help. He had had only one case of the kind, and that patient died; and he found post mortem that nothing was draining through the silver-wire drain; it was found to be quite choked.

Dr. DUNDAS GRANT said Dr. McKenzie, in this case, had carried out a treatment which was advocated as the most beneficial at a meeting of the German Otological Society, in the discussion as to the curability of otitic purulent meningitis. It was considered that the best results followed incision of the dura mater, and the exhibitor's admirable result seemed to have confirmed the view arrived at there.

Dr. DAN MCKENZIE, in reply, said that since the practice of draining the meninges had been started, there had been a larger percentage of recoveries, and that in itself justified the practice. He concluded that the dural incision was sufficient. There was no lateral sinus disease. He destroyed the labyrinth to drain the meninges. He had combined the two methods, believing that if the operator could open two doors instead of one, it was the right thing so to do.

**Microscopic Specimen of Cells from Cerebrospinal Fluid,
showing Organisms.**

By W. M. MOLLISON, M.C.

THE patient, a child, aged 6, was admitted to Guy's Hospital on account of drowsiness and a raised temperature, 101° F. There was a history of some discharge from the right ear, but there was almost none on admission. Lumbar puncture showed the fluid to be under increased pressure and cloudy. Apart from this sign of meningitis the patient exhibited no localising signs at all; she was drowsy, had a fluctuating temperature, and a pulse-rate corresponding to the temperature. Examination of the ear showed slight moisture on a disorganised membrane; this was not foul. Operation disclosed cholesteatoma in the antrum and a small temporo-sphenoidal abscess above the tegmen antri. The pus was very foul. Lumbar punctures were repeated each day for four days, but though the pressure was raised the fluid was not cloudy and in a week it had become normal.

Cases of Herpes Auris.

By W. M. MOLLISON, M.C.

Case I.—Man, aged 18, attended Guy's Hospital on account of a right-sided facial paralysis. Ten days previously he had had a blow on the head in the left parietal region. Following this he was in bed for four days with headache and sickness. The day after the blow he noticed facial paralysis. A few days later some spots appeared on the auricle; he had no pain in the ear, no deafness, no vertigo. The patient had a complete right-sided facial paralysis, and on the right auricle were scars of what had been herpetic spots; these scars occupied the concha and reached up along a line through the crus of the helix to the outer and upper edge of the auricle.

Case II.—The following case is a contrast to the foregoing: A soldier, aged 21, was seen at a Red Cross hospital with a view to operation for acute mastoiditis. Eight days previously he had noticed a lump in the

neck, had earache and was deaf in the right ear; some spots appeared on the auricle. He was three days in Chatham Hospital and was then transferred to the Red Cross hospital. He then was complaining of acute pain in the right ear, and had marked tenderness over the mastoid process. The pain was so bad as to keep him awake and was not relieved by doses of aspirin. These symptoms and signs, together with a swelling below the tip of the mastoid and a temperature of 100° F., suggested acute mastoiditis. On examination the right auricle presented a number of red scars on the external aspect, again fairly obviously the remains of herpetic spots; there was an enlarged gland below the tip of the mastoid. The tympanic membrane was normal, but the patient was very deaf (? totally so). The mastoid process was very tender, but this tenderness was more superficial than deep, more of the nature of a hyperæsthesia. Aspirin and small doses of quinine were prescribed. Three days later the patient was much better, but an additional interesting feature developed; he had, while walking in the garden, an attack of vertigo which caused him to fall.

DISCUSSION.

The PRESIDENT asked whether the facial palsy had disappeared in the second case; also whether the deafness had persisted. What would be the cause of deafness in a case of herpes? Herpes associated with disease of the geniculate ganglion was not usually accompanied by dullness of hearing. Conceivably there might be some inflammatory exudate affecting both the geniculate ganglion and the cochlear branch of the auditory nerve, travelling back and infecting the cochlea.

Mr. RICHARD LAKE said that years ago he showed a series of cases of herpes, and in all the membrane was affected; only one had herpetic spots besides. There were no spots on the membrane in these cases.

Dr. DAN MCKENZIE said that Ramsay Hunt had worked out the subject of herpes in relation to the geniculate ganglion. One or two cases had been shown some time ago; he believed Dr. H. J. Davis showed one. The deafness was due to implication of some of the other ganglia, and as a rule persisted. Dr. Pritchard had pointed out how closely these cases in the early stage resembled acute mastoiditis.

Mr. MOLLISON, in reply, said that when he first saw this case of herpes there was complete paralysis on the right side and the herpetic spots had almost disappeared, though there were still scars. He had seen four cases of herpes on the auricle and none had spots on the membrane. Some of them had facial paralysis, others had not. One of these present cases had it and the other

had not. The interest of these two cases was that they were completely different; in one case there was facial paralysis, herpes, and no deafness; in the other case herpes, no facial paralysis, but great deafness. Ramsay Hunt spoke of deafness and vertigo as a characteristic feature of some of these cases of herpes due to implication of other ganglia as well as the geniculate. He could not be sure as to the totality of this man's deafness, as he examined him at Westcliff, where he had not proper means of testing. In the case of the second patient, the doctor telephoned to say he was certain the mastoid must be opened, because of the swelling below the mastoid, the pain and the tenderness.

Case of so-called Primary Acute Mastoiditis.

By W. M. MOLLISON, M.C.

T. P., AGED 4, attended Guy's Hospital on April 20 on account of a swelling over the right mastoid process; the swelling was said to have been present four days. The child's mother said the right ear had ached ten days ago, but there had been no discharge. The right auricle was displaced downwards and forwards, and over the mastoid process was a red, fluctuating swelling, scarcely tender; the tympanic membrane was normal. Operation was performed and pus found in the mastoid process, and sticky pus in the antrum. Cultivation showed pure growth of pneumococcus.

DISCUSSION.

Mr. STUART-LOW said that during the last winter he had seen a number of these cases in which the middle ear and tympanic membrane were normal and the mastoid had become rapidly affected. In his opinion they were all secondary to throat implication and were influenzal in nature. Dr. Wyatt Wingrave had proved this by finding the influenza bacillus.

Mr. E. D. DAVIS asked whether Pfeiffer's bacillus was actually found in the cases spoken of by Mr. Stuart-Low, as a pneumococcal infection was often mistaken for an influenzal. Dr. Emery had said that there might be three organisms present in supposed clinical influenzal cases, Pfeiffer's bacillus, the *Micrococcus catarrhalis*, and the pneumococcus, or either of them, but that the bacillus of influenza was rarely found in these cases.

Dr. DUNDAS GRANT said it was agreed that even in cases diagnosed by competent people as influenzal it was rare to find Pfeiffer's bacillus; the bacteriology was mixed. He asked whether these anomalous ear and mastoid

cases were not almost confined to children. There was something special in the pathology of the child's ear. The antrum was comparatively large, as compared with the tympanum, and disease in the tympanum would settle down while it remained focused in the antrum. Tuberculosis would appear as a localised infection of the surface of the mastoid while the middle ear might be almost untouched.

Mr. NORMAN PATTERSON said that in one case on which he had been called to operate, and where the temperature was 104° F., the right ear appeared to be the culprit. He opened up the right mastoid and found some pus in the cells and antrum; the sinus was exposed and found to be normal. This was in the evening. The next morning the temperature had dropped to normal. But in the evening the house surgeon notified him that the patient had had a rigor. On again examining the left ear he could find nothing indicating disease. He, however, opened up the left mastoid, but beyond congestion discovered nothing. He thought it advisable to explore the posterior fossa, and on doing so found an extradural abscess and a thrombosed sinus. He could only suppose that the infection of the tympanum and antrum had taken place, but had cleared up owing to the possibility of drainage. On the other hand, infection having once reached the interior of the skull, and pus having once formed, the pus then became pent up. Drainage could not take place from this region.

Case in which Suppuration had performed a Radical Operation.

By W. M. MOLLISON, M.C.

W. H., AGED 14, was brought to Guy's Hospital in January, 1915. He had had left facial paralysis since he was 4 years old, caused perhaps by some incisions in the neck which were made when the boy was that age. There had been left otorrhœa for about the same time. The left ear shows a "radical" cavity, and there is no scar over the mastoid process.

Dr. DUNDAS GRANT said such cases had been shown before the Section, and he had described cases of cholesteatoma in which he found a homogeneous lining membrane in the cholesteatoma, which he thought well to leave in situ. He thought he had caught those cases just at the stage when the suppuration was going to perform a radical operation on its own account. He had not seen such cases recently, possibly because they were not allowed to go on so long.

Operation for Epithelioma of the Auricle with Secondary Involvement of Glands, May, 1912 (Three Years ago); no Recurrence. Specimen shown.

By NORMAN PATTERSON, F.R.C.S.

PATIENT, a male, aged 61, had a small growth on the pinna with well-marked enlargement of the cervical glands. There was a large hard mass over the upper part of the jugular vein, underneath the sternomastoid.

The operation consisted in removal of the auricle, together with a very free dissection of the neck. The internal jugular and also portions of the sternomastoid muscle and parotid gland were removed. Glands, fascia, &c., were taken away in one mass. In order to avoid trouble with the internal jugular in the upper part of its course, the lateral sinus was exposed early in the operation and a tampon of gauze placed between it and the skull wall, so as to cut off nearly all the blood entering from above.

(A full account of this case appears in the *Lancet*, April 5, 1913.)

DISCUSSION.

Dr. H. J. DAVIS said that he had exhibited an old man with epithelioma of the helix, and the case was published, with photographs, in the *Proceedings* in 1913.¹ He had removed the auricle completely and it was now in the College of Surgeons' Museum. He did nothing to the glands as none were visibly enlarged. There had been no recurrence. The patient, a gardener, was sensitive about his deformity until an artificial ear was made for him, which answered admirably.

The PRESIDENT said that usually in epithelioma of the auricle the glands were enlarged only late in the disease; it was one of the most favourable forms of epithelioma to deal with if seen fairly early.

Mr. PATTERSON replied that there was no doubt about the glandular enlargement in this case; it was very marked. But the primary growth was a comparatively small one.

¹ *Proc. Roy. Soc. Med.*, 1913, vi, p. 88.

Tympanic Membrane in Right Ear, moving with Respiration.

By DAN MCKENZIE, M.D.

THE patient was a woman, aged about 25. The affected membrane was thin and atrophied. Movement on respiration was clearly visible. The condition had been accidentally discovered by Dr. Ryan, one of the clinical assistants at the Central Throat and Ear Hospital.

DISCUSSION.

Dr. DUNDAS GRANT thought there must be some obstruction of the nose. When the patient inspired, there was more suction in the Eustachian tube than there would be if air passed freely through the nose.

Mr. STUART-LOW said that he had met with two well-marked cases of this condition in one month at his hospital clinic, and had shown them years ago at the British Laryngological Society. In his cases the Eustachian tube on the affected side was very patent and the tympanic membrane very thin and atrophic, and the hearing was considerably impaired.

Epithelioma of Auricle treated by Diathermy.

By DAN MCKENZIE, M.D.

MALE, aged 71. Epithelioma of auricle of two years' duration. (Microscopic examination by Dr. Wyatt Wingrave.) The growth involved a large surface of the auricle and had led to destruction of about one-third of the pinna. It had also extended to involve the mastoid region. No enlarged glands could be felt. There was severe pain.

Six weeks ago under chloroform the growth was treated by diathermy. The result had, so far, been satisfactory. The diseased tissue was apparently all removed and the ulcer which had formed was rapidly contracting and healing.

There had been no pain since the operation.

DISCUSSION.

Mr. STUART-LOW congratulated Dr. McKenzie on the seemingly good result in this case, but, of course, it was yet too soon to think that no recurrence would take place. He asked if the epithelioma was in an early stage and if there had been any enlarged glands and how they were treated. Mr. Stuart-Low said that he had originated the method of diathermy puncture of malignant glands and had shown cases at the Laryngological Society greatly benefited by it, and this method would be applicable to such cases as that now shown by Dr. McKenzie if enlarged indurated glands were present.

Dr. KELSON asked whether diathermy was not really cauterisation, and whether it possessed any definite advantages over Paquelin's cautery, excepting that it could be done deeply without necessarily damaging the surface to which it was applied.

Dr. DAN MCKENZIE replied that the ordinary cautery was applied to the surface of mucous membrane or of an ulcer. The cautery itself was hot, and charred the surface in contact, and the carbon formed there acted as a non-conductor of heat. Consequently the heat could not penetrate beneath the burned surface. But in diathermy the terminal did not get hot; it was the tissue that became hot over a certain surrounding zone. A broad terminal was placed in contact with the chest or back, and over that broad field the waves were scattered, but concentrated themselves upon the operated part where the terminal was punctate. Here a charred zone was produced, and beyond that, coagulation necrosis. In healing, the charred zone gave way first, then the coagulation necrosis slowly disappeared; this latter acted protectively against bacterial invasion. Cancer was not a surface condition, but an infiltrating disease; and if the therapeutic agent could be got to infiltrate along the same lines as the diseased cells were progressing, there was likely to be a better result than that obtained by simply charring the surface with the ordinary cautery.

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PATHOLOGICAL SECTION



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Pathological Section.

December 1, 1914.

Dr. F. W. ANDREWES, President of the Section, in the Chair.

Wound Inoculation in Carcinoma ; with Experiments upon the Action of Local Cytocides as a Means of dealing with it.

By S. G. SHATTOCK and L. S. DUDGEON.

De Liquoribus ad Cellulas destruendas aptis supra Vulnera chirurgica sparsas in operibus quibusdam Tumores adversum malignos.

SUMMARIUM.

VULNERA chirurgica nonnunquam cellulis infici in tumoribus malignis removendis bene cognoscuntur.

Sic accidit aliquando in pharyngectomiâ laterali dum tumor per incisionem externam extrahitur ; et in organis removendis si capsulae eorum ruptae sint.

De coli carcinomate in mente oportet, insuper, haberi epithelii cellulas a superficie naturaliter abradi ; et quum actiones aut pepticae aut trypticae in colo absint, has cellulas in tumore excidendo vulnus chirurgicum inficere posse.

Tumoris cujusque maligni membranaeque mucosae circumjacentis superficies ob eandem rationem sterilizari debent contra actionem non solum bacteriorum sed etiam cellularum malignarum.

In experimentis nostris tumoribus inurium malignis usi sumus (Carcinoma numero 63, Imperial Cancer Research).

Mure interfecto neoplasma excisum est et telâ ejus connexivâ extractâ, tumoris pulpa cum liquore quodam selecto commixta est.

2 Shattock & Dudgeon: *Wound Inoculation in Carcinoma*

Liquore denique exhausto pulpa sub murium cute sanorum inserta est ut monstraretur utrum tumor oriturus esset an cellulæ malignæ mortuæ essent.

Liquores cum quibus experti sumus erant: Aqua destillata; hydrargyrum colloide; selenium colloide; liquor iodi, 1:100; liquor iodi, 2:100; liquor hydrargyri perchloridi, 1:1,000, 1:500, 1:600; liquor zinci sulphatis, 1:240; hydrogen peroxidum; aqua radii; liquor sodæ taurocholatis, 2:100; alcohol absolutum.

Liquoris actio post horæ duodecimam partem terminata est aquâ destillatâ substituendâ.

Hæc observationes demonstrant nec aquam destillatam nec hydrargyrum colloide nec selenium colloide nec hydrogen peroxidum nec aquam radii nec liquorem sodæ taurocholatis ad cellulas destruendas utilia esse.

Liquor zinci sulphatis, 1:240; liquor iodi, 1:100 aut 2:100; liquor hydrargyri perchloridi, 1:500 aut 1:600; alcohol absolutum; hæc omnia tumorem augeri prorsus prohibent.

With the subject of wound infection that of auto-inoculation is so closely connected that before taking up the first we may refer to the latter phenomenon, as well as briefly to the kindred topics of the heredity of carcinoma, and the theoretical possibilities of its being an infectious and contagious disease.

Heredity.

That carcinoma is to some extent hereditary or familial is not only established by the analytical method of clinical observation, but it has been confirmed by the synthetical investigation of Dr. J. A. Murray, carried out at the Imperial Cancer Research.¹ This method consisted in breeding large numbers of mice from such as suffered from spontaneous cancer, crossing their offspring in the most varied manner, and recording all the facts as to age, sex, and cause of death in the animals so obtained throughout a period of years. From the accumulated data it is possible to construct complete pedigrees, showing the frequency and position of malignant new growths in the ancestry of each. It appears from these data that the disease was more frequent in those

¹ The clinical evidence upon this subject is well set out by Mr. Roger Williams in his work "The Natural History of Cancer," 1908, p. 356, to which work we are indebted for certain of the references cited in the present communication.

cases where the mother, or one or both grandmothers, or all three, suffered from cancer. Dr. Murray concludes that an hereditary factor, the influence of which can be concentrated by selective mating, plays an important part in determining the frequency with which stocks of mice spontaneously develop malignant neoplasms.¹

In regard to *grafted* mouse cancer it does not appear, from Haaland's experiments,² that mice from which spontaneously growing carcinomata have been excised give a greater percentage of positive results than normal ones.

The fact that malignant tumours may be congenital—i.e., present at the time of birth—is in itself a proof, moreover, that they may be inherited. But until the essential cause of carcinoma is known, what it is that is transmitted will not bear defining.

If we take the ability to predict, as the test of accuracy in knowledge, it will be obvious, again, how remote is the possibility of any practical application of eugenics for the extinction of the disease. The scientific regulation of marriage for the purpose of prophylaxis would involve, in any given case, a knowledge of pedigree so distant and precise that the thing is beyond the credibility of realisation. And before the advent of such a consummation we may hope that cancer will admit of cure.

Infectiousness.

A transference of carcinoma might conceivably be brought about either by means of a free parasite or by the cancer cell in a living state, whether the biological peculiarity of the latter were due to a symbiotic ultramicroscopic parasite or not. We do not propose to reopen here any discussion on the theoretical possibility of carcinoma being a parasitic disease—not in the sense that the cell *per se* is a parasite, but that the invasive character of the cell is due to its being infected with an extraneous parasite; with an ultra-microscopic symbiote. It may be observed, nevertheless, that the various facts established in regard to immunisation and prophylaxis in mouse cancer do not absolutely disprove the presence of such a parasite, since they deal only with the destruction of the cell, and without the susceptible cell the parasite might be rendered inoperative.

Of the first conceivable method of transference nothing can be said,

¹ J. A. Murray, "Studies on the Heredity of Cancer in Mice," *Internat. Congr. of Med.*, Lond., 1913, Section of General Pathology and Pathological Anatomy, pt. ii, p. 67.

² Imperial Cancer Research: Fourth Scientific Report, 1911, p. 71.

4 Shattock & Dudgeon: *Wound Inoculation in Carcinoma*

for nothing is known of the hypothetical parasite—so far, at least, as carcinoma is concerned. In regard to the second, if the living carcinoma cell (whether the carrier of an ultramicroscopic contagium or not) is substituted for a pathogenic micro-organism, the hypothetical possibilities of its transference and implantation run parallel. Cancer cells may be freed and distributed from growths situated on external parts: they may be expectorated or otherwise voided by the mouth in carcinomatous disease of the tongue, lip, or larynx; they are passed in the urine in cases of renal or vesical carcinoma, and by the intestine in carcinomatous disease of the colon and rectum. In the wider, pathological use of the term, carcinoma is an *infective* disease, since, starting at a single spot, it invades the circumjacent structures, and may produce secondary distant lesions.

Whether the disease is *infectious* in the more restricted clinical sense—i.e., conveyable from one individual to a second without personal contact—is still a question that cannot yet afford to be ignored. Guelliot,¹ in an analysis of seventy-seven cases in which a transference suggested itself, points out that seventy-one were man and wife; and six, persons living together—masters and servants, parents, &c. In nineteen of this total the disease affected similar organs (stomach eleven, mouth four, skin two). In fifty-eight it affected dissimilar parts. It has been urged by some that the latter fact disposes of the possibility of the disease having been transferred. This is not, however, the case. The result can be viewed as due, not to the implantation of the living cells under such circumstances, but to the access and different distribution, “direct” or “indirect,” of a hypothetical symbiote; the cell may be dead without the virus being so, or the virus may have been liberated.

One factor which tends to prevent the success of an air-borne implantation of the carcinoma cell, whether into the lung or the more accessible higher parts of the respiratory track—the mouth, nasal cavities, or larynx—is the effect of drying upon the cell; this effect, as we have shown by experiments upon mouse cancer, is lethal.² This alone places the problem on a different basis from that of an air-borne pulmonary infection—say, with the tubercle bacillus. And as a matter of observation, primary carcinoma of the pulmonary parenchyma is one of the rarest of lesions, the recorded cases being particularly few. We have never ourselves seen an example of it, and there is none in the Museum of the Royal College of Surgeons.

¹ *Gazette des Hôpitaux*, 1892, lxxv, p. 1209.

² S. G. Shattock and L. S. Dudgeon, *Proc. Roy. Soc. Med.*, 1912, iv (Path. Sect.), p. 73.

As regards the possibility of infection by cell implantation from without, occurring in the course of the alimentary track: Many years ago one of us (S. G. S.), in conjunction with Mr. C. A. Ballance, fed white rats for prolonged periods with recently excised human mammary carcinomata. The animals ate the material with avidity, but no infection ensued. As this experiment involves a heteroplastic grafting, no positive result (as one now sees) could have been expected, for human carcinoma, say of the breast, will not grow when transplanted beneath the skin, or into the abdominal cavity of the lower animals (monkey, dog, cat, rabbit, sheep); the grafts undergo coagulation necrosis, and either become encapsulated or removed by phagocytosis.¹ We ourselves have obtained no more positive results after sensitising the animals with the blood of human patients suffering from carcinoma.

In June, 1913, we sensitised two rabbits, each with a single large intraperitoneal injection of the blood withdrawn into sodium citrate salt solution, from patients suffering with inoperable carcinoma of the breast. For each animal 30 c.c. of blood in 100 c.c. of citrated saline were used, the blood being obtained from two different patients, and kept distinct. Twenty-six days later a suspension of human cancer juice, obtained by cutting a mammary carcinoma, just excised, into thin slices and scraping the divided surfaces, was prepared. This was injected, in the case of one rabbit, into the auricular vein; in the case of the second, into the peritoneum. The animals showed no signs of illness at any time. They were killed five weeks later; no trace of any growth was discovered in the peritoneal cavity, or in the lungs or other viscera. To this negative result may be added a further one obtained in an experiment to ascertain whether human carcinoma might be successfully grafted into the rabbit after the preparation of this animal with the blood of another rabbit which had received injections of normal human blood.

These experiments were undertaken on the ground of certain results obtained in an investigation on the lytic action of the blood of animals which had been previously treated with the blood of another species.² This work showed that the repeated intraperitoneal injection of guinea-pig's blood into rabbits led to the production not only of a hæmotoxin or hæmolysin, specific for the blood of the guinea-pig, but of cytotoxins

¹ C. A. Ballance and S. G. Shattock, *Proc. Roy. Soc.*, 1890, xlviii, p. 392.

² "The Action of Splenotoxic and Hæmolytic Sera on the Blood and Tissues," L. S. Dudgeon, P. W. Panton, and E. Athole Ross, *Proc. Roy. Soc. Med.*, 1909, ii (Path. Sect.), p. 64.

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which acted notably upon the epithelial cells of the liver and kidney—i.e., that the serum was polyvalent in its toxic or lytic properties, or contained a series of lysins specific for each of the tissues affected. These results indicate that the blood of the guinea-pig originally introduced intraperitoneally into the rabbit had led to the elaboration of this complex antibody by reason of its containing itself either a single, correspondingly complex, substance, or a series of substances corresponding with the several tissues above enumerated.

As extended to the procedure of grafting human carcinoma into the rabbit, the possible result may be shown in the following scheme: (1) Human blood to rabbit = leads to the formation of a polyvalent cytotoxin or lysin for human tissue. (2) Blood from rabbit so treated, injected into a second rabbit = leads to the formation of a polyvalent anticytotoxin or antilysin. (3) Human carcinoma grafted into the second rabbit = might be protected against destruction by the presence of the polyvalent antibodies already produced in its blood. In the experiments made, two rabbits received ten intraperitoneal injections, at about weekly intervals, of blood drawn from the ear of two other rabbits which had received periodic intraperitoneal injections of human blood and whose serum was hæmolytic to human red cells. Finely divided human scirrhus carcinoma, just excised, was afterwards inserted subcutaneously into the two rabbits just referred to, the intraperitoneal injection of rabbits' blood being repeated thirteen days later. Eight days later a second insertion of scirrhus carcinoma was made. This was followed by three further injections of rabbits' blood at intervals of six, fourteen, and seventeen days. The animals were killed thirty-six days and fifty-five days afterwards respectively. The grafted material was found loosely encapsulated in the subcutaneous tissue, and partially calcified after necrosis; it had undergone no increase in volume.

So far as feeding experiments are concerned, the only correct procedure would be to feed mice with mouse carcinoma; the grafting would then be isoplastic. On May 25, 1914, we commenced feeding four mice (not fully grown) with the tumours raised by grafting other mice. The tumour used was that known at the Imperial Cancer Research as No. 63, the original of which grew in the mammary gland, and for the strain of which we are indebted to the director, Dr. E. F. Bashford, whose ever-ready assistance and that of his colleagues we again gratefully acknowledge. Mice with growing tumours were killed at intervals, the tumours being excised and pinned to a slip of wood.

The animals were fed in addition with oats, carrot, &c. The tumours, which were eaten with readiness, numbered, between May 25, 1914, and December 31, 1914, thirty-three. During the course of the experiment, many litters of young were born. At the date last given the animals were in perfect condition and without any sign of disease. The digestive processes occurring in the stomach and small intestine would, one may believe, jeopardise the implantation of any such ingested material in a viable state. Yet in the tonsil, where no such destructive factor obtains, infection might arise from the lodgment of cells in its crypts.

Contagiousness.

Although the clinical evidence may be read to show that carcinoma is conveyable by contact, it must be admitted that it is so only to a very limited extent. The great test case is, of course, that of uterine carcinoma. Although this is so common an affection, the number of times it has been transferred to the male is small. In his work on surgical diseases of the penis, Demarquay¹ states that of 134 patients with carcinoma of this organ only one had a wife suffering from carcinoma of the uterus. Similar evidence is furnished by Bossi,² who found that of 180 husbands known to have had marital relations with wives suffering from uterine carcinoma, not one had acquired the disease. Against this, however, must be placed the statistics of Guelliot and of Behla. Guelliot (loc. cit.) collects twenty-three instances of penile carcinoma consecutive to carcinoma of the uterus. Among the total forty-three cases of the same concurrence collected by Behla and Guelliot there occurs one reported by Tross, in which the carcinoma of the penis was regarded as identical in structure with glandular carcinoma of the cervical canal. Certain of these cases would doubtless fall under rigid criticism; but allowing this, the conclusion indicated is that in rare cases an isoplastic form of grafting does take place under such circumstances. That such a form of implantation is *possible* is proved *ipso facto* by the experimental inoculation of mouse cancer into mice. But as in the latter case there are limitations, so in the other. Some carcinomatous neoplasms arising spontaneously in mice are readily transferable, others with difficulty, and some not at all; and the second factor is the susceptibility of the animals inoculated, of which some readily take the disease and others are refractory.

¹ "Mal. Chir. du Pénis," 1876.

² *Gaz. degli Osped.*, 1902, xxiii, p. 378.

Auto-inoculation.

When we come to the question of auto-inoculation, or autoplasmic grafting, the case is different. The phenomenon of metastasis, whether by lymph or blood-stream, is, of course, the most common example of it. And it is to the point to observe that the transported cells under such circumstances grow at first without a blood supply, as they might *in vitro* in an homologous plasma. The whole process has been experimentally studied in the rat by Dr. J. A. Murray, at the Imperial Cancer Research. A suspension of rat sarcoma in salt solution was injected into one of the caudal veins of a second rat. Tumours grew in the lungs without any local infection arising at the site of operation.¹ The vascularisation of the growing embolus is a secondary phenomenon. But in ordinary speaking the term "auto-inoculation" is usually limited to the inoculation of free surfaces occurring in the same individual.

The simplest example is obviously that in which the transference takes place between two surfaces actually in contact: "contact carcinoma." And it is surely of much historical interest that the first recorded observation upon this was made by John Hunter. The case is well worthy, therefore, of being referred to in detail. The specimen, now in the Museum of the Royal College of Surgeons (Series of General Pathology: Biology of Neoplasms) consists of a prominent, somewhat pedunculated tumour which was removed from the perineum. The following quotation from the Hunterian MS. is headed "Cancer": "Mrs. Elliott, two years ago, had a small excrescence on the right side of the perineum, close to the verge of the vagina. This was allowed to increase for two years before she applied for any relief, and then it was about the size of a common egg; but by this time it had contaminated the parts that had been continually in contact with it, which were the back end of the right labium, both externally and internally, with the inside of the left labium and the angle uniting the two. The upper of these parts was a surface that rose a little higher than the skin, a good deal like granulations, but in some places more warty. As the surfaces seemed evidently to have become diseased in consequence of being in contact with the others, it was imagined if the others were removed these might get well of their own accord, or at least with little trouble. Several escharotics were used, but to no

¹ One of the specimens illustrating this is preserved in the Museum of the Royal College of Surgeons, in the series, "Biology of Neoplasms."

purpose, and then it was thought advisable to extirpate the original tumour wholly by the root. Very little change for the better took place in the diseased surfaces; inguinal glands became affected, and the different tumours increased; sloughing occurred in various degrees, and ligatures were placed on some. The health improved, but death occurred suddenly with difficulty of breathing."

The histological examination of this tumour (S. G. S.) shows it to be a papillary squamous-celled carcinoma. The epithelium presents an abundance of mitoses, and cells of great size with closely apposed multiple nuclei, the whole appearance being one of active proliferation; nests are scanty and small. The cell groups towards the surface are provided with a peripheral palisade series, which is absent in the deeper; none of them exhibit central keratinisation. The connective tissue around the invading processes is the seat of small-celled infiltration.

The deepest part of the fibrous tissue forming the stalk is free of invasion. This is further evidence that the surrounding growths were due to inoculation and not to continuous extension. The pedunculation indicates that the neoplasm was originally either a simple papilloma or a soft fibroma, in which malignant transformation subsequently took place.

In Hunter's account there is everything but the word "auto-inoculation"; and after all, perhaps, "contamination," which he uses, is etymologically the more correct. It is in this position, indeed, that the occurrence of contact carcinoma has been most frequently observed, and is best authenticated. Sir Henry Butlin¹ has referred to four such instances, in which the bilateral growths were microscopically shown to be carcinomatous, and where extension by continuity was excluded. In the College Museum there is a further example, of which the following is the description: The external generative organs of a woman removed during life. The posterior portion of the right labium majus is the seat of an extensive carcinomatous ulcer 6.5 cm. ($2\frac{1}{2}$ in.) in length and 3 cm. ($1\frac{1}{4}$ in.) in breadth, which involves also the posterior margin of the right labium minus. On the deeper, inner aspect of the left labium there is a second ulcerated carcinoma about 2 cm. ($\frac{3}{4}$ in.) in length, which is quite distinct from the larger and older lesion. The parts have been cut across in the mid-line behind for the purpose of removing a complete slice for microscopical examination. The histological sections revealed

¹ "Address in Surgery," British Medical Association meeting, August 3, 1907, *Brit. Med. Journ.*, 1907, ii, p. 255. The four instances occurred in the practice of Dr. Griffith, Mr. Bruce Clarke, Miss Aldrich-Blake, and Dr. Cuthbert Lockyer.

no epithelial extensions in the connective tissue. The parts, together with the lymphatic glands and fat in both groins, were excised from a widow, aged 56, the mother of four children. Menstruation had ceased for five years. The patient had complained of pruritus prior to the development of a growth the size of a pea on the right labium; the tumour gradually enlarged, causing pain, and being accompanied with discharge. The disease had been in progress for about six months. Among continental observers Hamburger,¹ Walter and others have put on record similar cases.

It can hardly be doubted that a similar process of direct inoculation obtains between the glans penis and the prepuce as a mode of extension superadded to that by continuity, so universally diseased may we find the two mucosal surfaces. There are striking examples of such in the College Museum:—

4470: The end of a penis removed by amputation, the prepuce being everted after having been recently slit up from the under side. On its inner aspect the prepuce is everywhere thickened from the growth of a squamous-celled carcinomatous neoplasm, the surface of which is very irregular as a result of ulceration and necrosis. The whole of the glans presents a similarly uneven and diseased surface. The patient died a year and a half after the operation, with secondary disease of the inguinal glands.

4463 is a similar specimen, in which the disease is equally extensive.

It is stated by Guelliot (*loc. cit.*) that Velpeau observed five cases of invasion of the glans at the site of contact with a diseased prepuce.

Examples of contact implantation from the uterus to the vagina are cited by Roger Williams (*loc. cit.*) from Thorn, Czempin, Fischer, and Russell. One of Thorn's² patients, a multipara, aged 46, whose uterus had been deflected to the right, where it had become fixed in its faulty position by adhesions, subsequently developed cancerous 'cauliflower excrescence' of the portio. Owing to the faulty position of the uterus, the diseased part was constantly in contact with the left side of the vagina, at a considerable distance from the fornix. In this position a papillary cancer developed which exactly fitted the similar growth on the portio. Elsewhere the vagina was free from disease. Parallel examples of contact infection between the cervix uteri and the vagina are brought forward by Velpeau, Hègar, Spiegelberg (*Guelliot, loc. cit.*).

¹ *Med. Chir. Rundschau*, 1892, No. 12.

² *Cent. f. Gyn.*, 1894, xviii, p. 228.

Of the three microscopically attested instances of contact carcinoma of the larynx adduced by Sir Henry Butlin (*loc. cit.*), one, David Newman's, concerned the ventricular bands, the other two the cords. One of these was observed by Sir Henry Butlin; the other occurred in the practice of Sir Felix Semon, and was microscopically investigated by one of us (S. G. S.).¹ Mr. Wilfrid Trotter informs us that he has seen three cases in which carcinoma of the epiglottis was associated with a localised secondary infection of the posterior wall of the pharynx; and Sir Frederic Eve tells us that he has seen a case where carcinoma of the posterior part of the alveolar border of the right side of the lower jaw was accompanied with a similar lesion of the apposed portion of the alveolar border of the upper. A like instance of contact infection between the jaws, which occurred in the practice of Mr. C. Stonham, is mentioned by Sir Henry Butlin (*loc. cit.*). Lücke² refers briefly to a case of ulcerating carcinoma of the border of the tongue, in which a contact implantation took place on to the mucosa of the cheek; other examples of this have been noticed by Guelliot, Hyvert, and Fenwick.

Of contact carcinoma as between the lower and upper lip, Von Bergmann³ recounts an excellent example in a man, in whom a tumour had been present in the middle of the lower lip for three months, and a second growth in a corresponding spot on the upper for only five weeks. Both tumours proved, microscopically, to be squamous-celled carcinomata, as stated by the author in answer to an inquiry of Sir Henry Butlin. In the discussion upon this occasion Hahn observed that Billroth had seen a similar case.

In connexion with the skin the two following instances are of interest. Mr. Harrison Cripps has referred to a case (observed at St. Bartholomew's) of auto-inoculation of the skin over the inner condyle of the humerus, where the arm had been closely bandaged to the chest, and kept for two months in contact with an ulcerating carcinoma of the breast;⁴ and Mr. De Morgan, to one of carcinomatous infection of a patch of the skin beneath an overlying pendulous breast affected with an ulcerating carcinoma, the lesions being separated by intervening unaffected skin.⁵ The multiple nodules of the skin, which appear

¹ *Path. Soc. Trans.*, 1888, xxxix, p. 36.

² Pitha and Billroth's *Handbuch*, Bd. ii, p. 50.

³ *Berl. klin. Wochenschr.*, 1887, p. 891.

⁴ *Brit. Med. Journ.*, 1882, i, p. 653; and again in the *Trans. Path. Soc. Lond.*, 1881, xxxii, p. 111.

⁵ "On the Origin of Cancer," 1872.

after the cutis overlying a mammary carcinoma has been completely infiltrated, and the investing epidermis abraded, have, conceivably, in part, a similar origin, and may result from the implantation of cells into hair-follicles or sweat-ducts, on a surface kept moist beneath dressings improperly used. And lastly, under the title of auto-inoculation of melanotic carcinoma of the eyelid, a case has been recorded by Vennemann,¹ but the original we have been unable to find in any London library.

These are cases of infection by direct contact, and they naturally lead on to those where a free surface is infected at a more distant spot by cell transference. Improbable as it might at first seem, such an occurrence has been observed in connexion with the skin. Sir Henry Butlin (*loc. cit.*) cites the history of a gentleman, under the care of Sir William Macewen, with a carcinoma of the lower lip, who acquired a trick of pressing the lesion with the finger and thereupon rubbing the tip of his nose. The tumour of the lip was excised, and three months later an ulcerating lesion appeared on the nose; this, when removed, proved to be a squamous-celled carcinoma, like that of the lip. Here it may be inferred that cells were translated from the labial lesion to patulous hair-follicles, wherein finding a suitable temperature and sufficient pabulum from the sebaceous secretion, they started growing, and then as a result of their mysterious biological faculty, commenced to invade the perifollicular connective tissue. The products of cell autolysis are known to be highly nutritive, those of muscle particularly so: in the hair-follicle the transplanted elements would be able to utilise those of the cells of the sebaceous glands. Schimmelbusch recounts the case of a man who was in the habit of handling his carcinomatous ear, and transmitted the disease to his lip through the medium of his finger-nails. Of the several cases recorded of multiple squamous-celled carcinomata of the skin, and of multiple rodent ulcers, it is not easy to say in how many the lesions are independent, and in how many they may have resulted from some similar method of auto-inoculation. Thus Mr. Jonathan Hutchinson, jun., has related that he operated upon an old man for carcinoma of the lower lip and of the back of the right hand, both of which proved to be of the squamous-celled variety; but though auto-inoculation is here probable, nothing was recorded as to the patient having been in the habit of rubbing his lip with the back of the hand, nor as to which growth was the first to appear.

¹ *Ann. Soc. Belge de Chir.*, Brux., 1895-96, iii, pp. 130-132.

From skin to mucosa, one example of transference authenticated by microscopical examination after death is that of an ulcerating squamous-celled lesion of the back of the right hand in a woman, aged 81, who was in the habit of rubbing the right eye with the affected part, and in whom the conjunctiva became secondarily affected—a literal inoculation of the disease. This interesting case is recounted by Kaufmann.¹ But the more numerous cases are those where the transference takes place from one part of a mucous track to another more distant. In the Museum of St. Thomas's Hospital there is a specimen of carcinoma of the œsophagus (917A), connected with which there is an interesting history.

917A: A larynx with the trachea, œsophagus, and a small portion of the stomach. The walls of the œsophagus, a short distance from its upper end, are extensively infiltrated with a carcinomatous growth which had led to marked stenosis. From the main growth long extensions of the disease are traceable in the mucous membrane; these are limited to the summits of the rugæ. The parts were obtained from a man, aged 38, who began to have difficulty in swallowing about six months before admission. Four days after admission an œsophageal bougie, the size of a No. 5 English catheter, was passed through the stricture, which was found to be situated 9 in. from the teeth. The tube was left in, and the patient fed through it during the day. From April 7 to June 28, 1891, he was treated by catheterisation, the instrument being left *in situ* for three days at a time or longer, and then changed for a larger one when possible. After a while the bougie could be dispensed with, and he was able to swallow minced meat. The dysphagia, however, returned, and the bougie was again used, though its size had to be diminished. At length, when only a No. 5 could be passed, gastrostomy was performed. Death occurred a fortnight later. The distribution of the growth here suggests that a local extension resulted from direct inoculation due to the use of the catheter.

In the College Museum there is a specimen of villous carcinoma of the œsophagus (1231A), in which a similar extension along the rugæ is accompanied with duplication of the growth, but unfortunately no history is recorded with it.

In the following specimen the presence of minute carcinomatous lesions in the neighbourhood of a diseased œsophageal ruga is associated with carcinoma of the cardia, and may be viewed as due to the displacement of cells from the latter into the adjacent part of the canal:—

¹ Virchow's Archiv, Bd. lxxv. p. 317.

2322B: The cardiac portion of a stomach, with the lower end of the œsophagus. The former is the seat of a carcinomatous elevation of wide extent, which microscopic examination shows to be of the spheroidal-celled variety. From one spot at its upper border there extends upwards a linear process 10 cm. (4 in.) in length, which apparently involves one of the rugæ; the surface of this is, for the most part, ulcerated. In addition to this there is a circular ulcer 0.7 cm. ($\frac{3}{10}$ in.) in diameter close above the cardia; and a close inspection of the neighbouring mucosa above this will show the presence of three or four minute, flattened elevations which, like some of those in the course of the infected ruga, are secondary carcinomatous foci, the presence of which may be explained (like the disease of the ruga itself) as due to the auto-inoculation of the mucosa by cancer cells, which have found their way into the lumen of the canal from the growth in the stomach. From a man, aged 60, whose illness began two years previously with pain and vomiting after food. The patient gradually became anæmic and emaciated, and shortly before death œdema of the legs and scrotum supervened. No metastases were found after death in the viscera.

Klebs¹ furnishes three striking examples of squamous-celled carcinoma of the stomach accompanying similar growths in higher parts of the alimentary tract covered with squamous epithelium. In one there were two large flat knots lying under the cardia near an ulcerating epithelioma of the œsophagus. In a second case, that of a man with an extensive epithelioma of the face, involving the mouth, throat and antrum, there was a knot of the same structure in the greater curvature of the stomach. In the third, a case of a large epithelioma on the dorsum of the tongue, there were also five or six round knots on the greater curvature of the stomach, having the same squamous-celled structure. Fütterer² has published another case of the same nature and refers to six further; and one from the tongue to the œsophagus is noticed by Ricard. Kraske³ has described two cases of high rectal carcinoma associated with small knots in the mucosa close by the anus; in both the secondary knots were, like the primary tumours, of the columnar-celled kind; but in the second case the nodule was seated on a spot invested with squamous-celled epithelium, whence the author concludes that the metastasis was due to a process of direct grafting, and that the first case allowed of a similar explanation.

¹ *Handb. der Path. Anat.*, i, p. 190.

² " *Medecine*," 1902, viii, p. 177.

³ *Centralbl. f. Chirurgie*, 1884, Bd. ii, p. 801.

Genito-urinary System.

Two examples of auto-inoculation affecting parts of the genito-urinary system are contained in the College Museum, and are of sufficient interest to cite. In one, a carcinoma of the bladder (3700A), the whole of the urethra (male) is infected. In the new edition of the College catalogue it is described as follows: "A urinary bladder laid open from the front. The posterior wall is the seat of an extensive carcinomatous growth which takes the form of lobulated projections, the surfaces of which have undergone ulceration. The disease has completely destroyed the mucosa and muscular wall over a large circular area, the destruction having been followed by a low hernial protrusion of the peritoneal investment; the disease involves, moreover, the prostatic portion of the urethra, which was widely dilated by portions of the neoplasm removed at the operation described in the history. The orifice of the right ureter, into which a piece of coloured glass is passed, is free; that of the left is involved in the growth. Microscopic examination of the neoplasm within the muscular wall shows it to be a carcinoma, in some places of the spheroidal-celled variety; in others, the association of columnar cells with spheroidal is indicative of its source from the transitional vesical epithelium. There are nowhere any cell nests. From a man, aged 48, who was admitted for hæmaturia accompanied with difficulty of micturition. His illness began six months previously with hæmaturia, but until six weeks before admission he was able to work as a farm labourer. He was at that date once sounded, as he complained of pain across the lower part of the abdomen. When admitted, the bladder was found distended, resistant, and tender; the passage of a sound caused violent expulsive efforts. The urine contained much blood and many clots. In order to drain the bladder a perineal incision was made, but before doing this it was observed that by compression of the urethra in the perineum a mass of new growth became extruded from the external meatus. On opening the bladder from the perineum a large tumour was found attached to the left side, around the ureteric orifice. The viscus was distended with the new growth, the pedicle of which was torn through and the mass removed. Cellulitis supervened and the patient died three weeks later. At the autopsy the left kidney was found to be the seat of suppurative pyelonephritis. The liver, lungs, and mediastinal glands contained metastatic growths. When the bladder was opened its cavity was found nearly filled with a sloughing growth, the greater part of which was washed

off with a gentle stream of water, leaving an ulcerated base; the growth extended into the track of the perineal incision.¹

In the female a parallel occurrence has been observed, in a case where, subsequently to the removal of a villous carcinoma of the bladder, a growth was excised from the urethra near the external meatus. On the bladder being reopened an extensive infiltrating recurrence was found in that viscus.²

The second specimen in the College Museum (3584A) is a kidney of which the pelvis and calyces are the seats of villous carcinoma. In two situations there are areas on the exterior where the growth is exposed; the larger of these represents a portion laid bare by the surgical division of the dilated pelvis. This exposure probably explains the recurrence which took place at the site of operation, the wound having been, as a result, directly infected by cells detached from the neoplasm. From a man, aged 70, who had suffered from painless hæmaturia for four years. The hæmorrhage had been constant for the last two years, and the patient was extremely anæmic. On cystoscopic examination swirls of blood-stained urine were seen issuing from the right ureter. The kidney was therefore cut down upon for exploration, but it was not opened nor removed, and the wound was closed. The hæmaturia persisted, and two years later, the cystoscope showing hæmorrhage from the right ureter, the kidney was removed. The hæmorrhage at once ceased, and in a few weeks the patient was about. A year later he returned with hæmaturia. Cystoscopic examination again revealed blood coming from the right ureter. On exploring the latter its upper end was found distended and globular from growth within; the lower end below the brim of the pelvis was unaffected. It was therefore resected as far as where it crosses the iliac vessels. A large cyst containing blood subsequently formed in the scar of the nephrectomy; this was excised; its wall was reported to be infiltrated with narrow branching processes of "squamous-celled" carcinoma. A small secondary nodule was afterwards detected in the prostate; this may have resulted from the implantation of cells in the ducts. Death occurred five years after the nephrectomy.³

¹ C. J. Symonds, *Trans. Path. Soc.*, 1894, xlv, p. 98; and J. H. Targett, *ibid.*, 1896, xlvii, p. 155, Case 3.

² Hartmann and Lecène, *Ann. de Gyn. et d'Obst.*, 1907, iv, p. 65.

³ E. Hurry Fenwick, "Ureteric Meatoscopy in Obscure Diseases of the Kidney," 1903, p. 81.

This example closely resembles one observed by Dr. Michels (Sir Henry Butlin, *loc. cit.*), where cystoscopic examination disclosed a papillary growth at the orifice of the left ureter, associated with a large tumour of the left kidney. The ureteric tumour was removed first, and as the hæmaturia persisted the kidney was, later on, excised. The kidney was found to contain a large papillary neoplasm. Both proved, on microscopic examination, to be malignant.

With respect to the uterus, cases of carcinoma of the body of the organ associated with the presence of similar disease in the cervix, and suggestive of infection by transference, have been recorded by Niebergall, Winter, Pfannenstiel, and Leopold. The most interesting of such a group is perhaps the following, where a columnar-celled carcinoma of the cervix uteri was found accompanied with a second ulcerated lesion on the right postero-lateral wall of the vagina which, on being excised with the uterus, proved to be of the same columnar-celled variety as that of the cervix.¹

Pulmonary Infection.

Of examples of pulmonary infection from the upper respiratory passages we have the oft-cited case, observed by Moxon,² of a man suffering from carcinoma of the œsophagus which involved the trachea; in the latter there were two low elevations, which did not, however, exhibit any abrasion or ulceration. After death the only secondary disease discovered was in the lungs, in the lower lobe of each of which there were several small rounded growths, not at the surface (as in ordinary metastasis) but in the centre of the lobules; small bronchia could be traced up to the lesions. Kraske (*loc. cit.*) gives a reference to a published case of carcinoma of the œsophagus, which perforated the trachea, and in which secondary growths were in a similar way found in the lower lobes of both lungs.

In one case³ where squamous-celled carcinoma of the lung was found combined with carcinoma of the larynx, the authors state that they had reason to believe that the infection resulted from the inspiration of material from the laryngeal lesion. Sir Rickman Godlee⁴ has

¹ Hartmann and Lecène, *Ann. de Gyn. et d'Obst.*, 1907, iv, p. 65.

² *Trans. Path. Soc.*, 1869, xx, p. 28.

³ Wilks and Moxon, "Pathological Anatomy," 3rd ed., 1889.

⁴ *Trans. Path. Soc.*, 1876, xxvii, p. 253.

recorded the case of a patient from whom an ulcerating carcinoma was removed from the left side of the tongue, partly by means of the galvanic *écraseur*, partly by means of scissors; extensive hæmorrhage occurred from the ranine artery, and as this threatened to produce suffocation, tracheotomy was performed, and the blood sucked out of the chest. There was no enlargement of the cervical lymphatic glands. The patient was readmitted twenty-two months later. Five weeks before this, chest symptoms had been complained of. After death a large squamous-celled carcinomatous mass was found involving the right lung, and there were two or three nodules in the left. No recurrence had taken place in the tongue, and there was no disease of the cervical glands. Striking as this case is, its value is lessened by the fact that sixteen weeks before the patient's readmission a squamous-celled carcinoma had appeared on the fourth toe, and in connexion with this the inguinal glands became secondarily diseased. Whether the growth on the toe were independent of that originally in the tongue or not, the lung *may* have become infected from the inguinal glands.

Auto-inoculation of Serous Membranes.

The Peritoneum.—The most notable example of this, although the disease is not carcinomatous, is the widespread peritoneal infection which follows the rupture of papilliferous cysts of the ovary. The lesions, as is well established, are not malignant, since they are not invasive; the transplanted epithelial cells shed from the intracystic growths incite the formation of secondary papillomata wherever they chance to become implanted. That peritoneal infection does not invariably follow the rupture of such cysts is shown by a specimen (No. 4498) in the College Museum: A portion of a large papilliferous ovarian cyst which ruptured several times and was eventually removed by Sir Spencer Wells. In the history of the preparation it is recorded that no intraperitoneal growths were found present at the operation. With this cyst is removed the distal $2\frac{1}{2}$ in. of the Fallopian tube. No implantation has occurred within this from any access of free intraperitoneal cells. Virchow's early observation that infection of the recto-vesical, recto-uterine, or utero-vesical pouches, and of other parts of the peritoneum, may ensue upon carcinoma of the stomach after the local involvement of the gastric peritoneum is well known,¹ and it is corroborated by clinical observation.

¹ *Krankhaften Geschwülste*, Lect. iii ("General Physiology of Tumours").

Mr. Wilfrid Trotter tells us that he has more than once been led to the diagnosis of carcinoma of the stomach by the detection of a ridge in Douglas's pouch on rectal examination. The recent extension of such an explanation to the bilateral infection of the ovaries, which may complicate carcinoma of the stomach, intestine, gall-bladder, adrenal, or mamma, needs more investigation than it has yet received. In regard to its association with mammary carcinoma, Mr. Sampson Handley would view it as due to a direct infection of the peritoneum in the mid-line at the epigastrium, resulting from a continuous extension of the primary disease, the ovarian metastasis taking place from this peritoneal focus. The peculiarity of the ovary may be borne in mind in this connexion. Unlike the testicle, it is covered with columnar, germinal epithelium, and it has no proper tunica albuginea distinct from the stroma. Even in the adult, moreover, primitive ova may occur amongst the columnar cells, and involutions or ingrowths may take place from the surface into the subjacent stroma. Whether the surface of the ovary merely presents vulnerable spots for such a process of carcinomatous implantation, or whether the germinal epithelium plays a selective and a biologically adjuvant part, are matters worthy of consideration and experiment. But, apart from this, certainly the normal rupture of the Graafian follicle exposes the interior in a peculiar way to the access of free carcinoma cells that may happen to lie on the exterior.

As a further extension of the same hypothesis Mr. B. Glendining¹ has endeavoured to show that the carcinoma of the Fallopian tubes, which may accompany the secondary carcinoma of the ovaries, is due to the entrance of shed cells through the abdominal ostium, and their implantation upon the tubal mucosa. Without doubt the converse is true—viz., that the peritoneum may be infected from the Fallopian tube. In the College Museum there is a specimen illustrative of this.

45841 : A left Fallopian tube, together with the ovary. The distal end of the tube for a distance of about 7 cm. ($2\frac{3}{4}$ in.) is considerably dilated by the growth of a carcinoma, which arises from the mucosa towards the ostium, and elsewhere projects in a free condition into the canal. The growth extends completely up to the abdominal ostium, which is slightly dilated and filled with it. The divided surfaces of the tumour present to the naked eye a complex papillary construction. On

¹ *Archives of the Middlesex Hospital*, 1910, xix, p. 68.

microscopic examination the neoplasm, although papillary in character, was found to have infiltrated the mucosa over the relatively limited area of its attachment near the ostium. From a sterile woman, aged 57, in whom a fibromyoma had for many years been slowly growing in the uterus; latterly, the menopause having ceased, irregular but frequent hæmorrhage occurred. The entire uterus with both the tubes and ovaries was removed. The diseased tube ran over the tumour of the uterus—a fibromyoma in its posterior wall, nearly 5 in. in diameter. The dilated ampulla of the tube rested in a peritoneal recess near the brim of the pelvis, by the side of the rectum. A process of the intratubal growth projected through the abdominal ostium, and had infected the adjacent peritoneum as far, at least, as the rectum.¹ The patient remained in good general health for eleven months, when signs of recurrence appeared in the pelvis, followed by death a few weeks later.

In the *pleura*, the striking extent of the growth in cases of plexiform sarcoma (“malignant endothelioma”) must be attributed in part to a similar process of auto-inoculation, for not only is the whole of the costal but the whole of the pulmonary pleura affected. When we reflect that the continuity of the two areas is limited to the line of reflection at the base of the lung, the cogency of such a view will be the more apparent. In the College Museum there is so typical an example of the generalised disease that it is worth citing.

1670: A vertical slice of portion of a right lung, and of the visceral and parietal pleuræ. Both layers of the membrane are greatly, and fairly uniformly, thickened from primary malignant disease. On the pulmonary aspect the growth has in varying degrees invaded the lung. Indications of the pleural cavity may be here and there recognised, occupied with a vacuolated coagulum of lymph. In the fat beyond the parietal layer there are many small local metastases. Inferiorly a portion of the diaphragm is shown adherent to, and involved in, the growth. Histological examination shows the neoplasm to be a plexiform sarcoma (“malignant endothelioma”). The individual cells composing the well-defined branching columns are throughout of spheroidal form. From a man, aged 21, who, two months previously, noticed a painful red swelling beneath the angle of the right scapula, which gradually increased in size. When admitted there were signs of effusion into the right pleura, and a hot, red, and tender swelling in the situation

¹ J. Bland-Sutton, *Trans. Obstet. Soc.*, 1902, xliv, p. 311.

first referred to. Ten ounces of blood-stained fluid were withdrawn, the swelling was cut into, and a piece removed during life for diagnosis. Death occurred about six weeks later. The left pleura and lung were normal. Some of the lymphatic glands around the cœliac axis were found to be secondarily affected.

In the production of this result it is not difficult to picture the inoculation of the surfaces as taking place by the distribution of detached cells over the pleural cavity brought about by the incessant movements of respiration; nor must the part possibly taken by the stomata of the serous membrane in intercepting such detached cells be lost sight of, nor the fact that the stomata themselves open into subjacent lymphatic vessels, by the permeation of which further extension would be facilitated. The cytological examination of pleural and peritoneal effusions accompanying malignant disease shows, moreover, that neoplastic cells are in some cases set free. In a concise summary upon cyto-diagnosis in general Dr. E. Athole Ross¹ points out that one of the first of such positive findings is that recorded by A. S. Warthin,² who diagnosed a primary sarcoma or malignant endothelioma of the pleura from an examination of the exudate. L. Gulland³ in an examination of the pleuritic fluid from three cases of carcinoma of the lung considered that cells derived from the growths were present. H. C. Earl,⁴ from a cytological examination of the effusions into serous cavities, concluded that carcinoma cells are occasionally undoubtedly present. E. A. Ross's own study in six cases of pleural and peritoneal effusions associated with sarcoma and carcinoma did not yield results of diagnostic value. Since that time, however, a positive diagnosis has been many times arrived at in the Clinical Laboratory at St. Thomas's Hospital, and the value of the method is now everywhere admitted.

To revert to the auto-inoculation of mucous membranes, the duplication or triplication of carcinoma of the large intestine receives, perhaps, its best explanation as being the result of such a process; and with this example we may close the list. It is a matter of clinical observation that a carcinoma of the cæcum is not infrequently accompanied with, or followed by, one of the pelvic colon.

¹ *Trans. Path. Soc.*, 1906, lvii, p. 361.

² *Med. News*, 1897, lxxi, p. 489.

³ *Scot. Med. and Surg. Journ.*, 1902, x, p. 490.

⁴ *Dublin Journ. of Med. Sci.*, 1903, cxvi, p. 409.

The most remarkable case of such multiplicity, probably, is the following, certain of the tumours from which are in the Museum of St. Thomas's Hospital. A gentleman was admitted into St. Thomas's Home under the care of Mr. C. S. Wallace, for a carcinoma of the transverse colon, which was removed in November, 1905. There was a two years' history of symptoms. In 1908 he was readmitted with a carcinoma of the sigmoid flexure, which was also removed. In 1911 he returned with a recurrence at the site of the first operation; this was likewise excised. In February, 1912, the same patient was admitted for the fourth time, with a carcinoma of the rectum, and this was in like manner removed.¹ In this case, as in similar ones, the inference is that the growths distal to the primary tumour have resulted from auto-inoculation; from the transport of cells from the surface of the neoplasm and their engagement in a distant crypt, in which they have commenced to multiply, and thence to invade the surrounding wall of the gut.

In the normal intestine, shed epithelium constitutes a not inconsiderable element of its contents. The older observation of L. Hermann, carried out upon an isolated loop of gut, has shown that the latter becomes filled at the end of some weeks with cellular debris, bacteria, and often fat, the whole having a fæcal appearance. Clinical observation confirms this. In cases where no food is being ingested by the mouth, and a patient is being fed with meat juice and salt solution *per rectum*, the amount of fæcal material passed will obviously exceed in volume the residue of what is introduced (Dr. Newton Pitt). More striking proof of the amount of epithelium shed from the intestinal mucosa is furnished by a microscopic examination of the contents of the normal vermiform appendix. Of a series of normal appendices pinned out and slit up under water, a fair number will be found to contain only a long, narrow, cylindrical plug of white, friable material, which on microscopic study, in dilute glycerine, will be seen to consist entirely of shed columnar epithelial cells. A microscopic study of the contents of the alimentary canal in the stillborn human fœtus yields, as might be anticipated, similar results. Thus the examination of the *gastric contents* of a full-term, well-nourished fœtus which died during birth showed large numbers of columnar epithelial cells lying in the tenacious mucus which separated the two surfaces; the contents were exposed by carefully slitting open and pinning out the stomach under

¹ C. S. Wallace, *Proc. Roy. Soc. Med.*, 1912 (Surg. Sect.), p. 143.

water. *Small intestine*: This contained a flattened, whitish, somewhat consistent ribbon impressed with the valvulae conniventes. Microscopically the material contained vast numbers of shed epithelial cells. On staining dried films with Sudan III a fair number of fine fat droplets were demonstrated, mostly free, but some within the epithelial cells. *Ascending colon*: The voluminous, viscous, coherent, yellowish-brown contents showed, besides vast numbers of fine orange-yellow granules of bile pigment and some cholesterin crystals, &c., small collections of flat epithelial cells in conspicuous numbers, and groups of fine hairs, the pigmentation and size of some of which proved that they came from the scalp. The presence of flattened cells is to be ascribed, like that of the hairs, to the foetus having swallowed lanugo and shed epidermis suspended in the liquor amnii, an occurrence now for some time recognised. *Vermiform appendix*: The contents of the appendix consisted of large numbers of shed epithelial cells, with a small amount of mucin; a few groups of minute orange-yellow particles of bile pigment indicated the access of meconium to the interior.

Now to apply these data to the case of intestinal carcinoma. We cannot doubt that from the free surface of a common papillary carcinoma of the colon a shedding of epithelial cells and their detachment by the passage of the firmer intestinal contents over the new growth, are things in constant progress. The ease with which such a detachment may be brought about is shown by the following simple observation: A typical infiltrating papillary carcinoma of the colon, just excised, was carefully slit up with a scalpel from without inwards on one side only, so as not to damage the interior, and the bowel pinned out; after the surface of the new growth had been gently washed with water a series of cover-glasses were lightly rubbed over some of its intact eminences without scraping them with the edges of the glass. A drop of dilute glycerine was then placed upon the smear (which had not been allowed to dry) and the preparations immediately examined. In each of the specimens plaques of well-conditioned carcinomatous epithelium were found. One of these is shown in the accompanying figure (fig. 1). This observation was confirmed upon two other recently excised carcinomata of the colon.

Have we realised the possibilities for evil which all this implies? For the cells shed or abraded from the intact surface of such a carcinoma are potentially adequate to reproduce the disease. Herein lies, as before observed, the best explanation of the duplication of carcinomata in the large intestine; and more than this, it may be an element in the

production of local recurrence at the site of excision. For even when the growth has been removed with its lymphatic extensions, should free, viable cells lying on the normal mucosa at the site of operation have been carried on to the face of the divided edges of the bowel, a local recurrence might be expected.

It is a significant fact in relation to the present subject, that whilst duplication of carcinoma of the large intestine is not so very uncommon, no instances are known in which a carcinoma of the stomach has been accompanied by one of the colon. How can this be explained? The reason may be found in the different character of the intestinal contents. Any viable cells detached from a gastric carcinoma, supposing that they escape digestion in the stomach, have to run the gauntlet of the small intestine, in which they could hardly escape tryptic digestion. In the large intestine, on the contrary, the digestive process is at an end, and cells might be transported in a viable condition from one spot to another, ready for implantation into a crypt or upon the face of a surgical wound. The presence of the ileo-cæcal sphincter—i.e., the sphincter at the extremity of the ileum, the existence of which was pointed out by Professor Arthur Keith (1903), and of which the physiological action was shortly afterwards investigated by Elliot—serves the distinct purpose of preventing the passage of chyme through the ileo-cæcal valve into the large intestine until the process of intestinal digestion is fully completed. Dr. A. F. Hertz¹ has recently shown that bismuth-containing chyme reaches the end of the ileum an hour or more before any appreciable quantity is allowed to pass into the cæcum. The chyme accumulates in the last few inches of the ileum, where it remains and undergoes digestion, actually for a longer period than in the stomach. The chyme which reaches the cæcum contains only a very small proportion of nutrient material in solution.

Lodged in a crypt, the nourishment of the transported cells would be ensured by its proper secretion, the succus entericus, which, whilst it has no digestive action, contains a certain amount of proteid. And once growth has been started the biological peculiarity of the carcinomatous epithelium would ensure the invasion of the wall of the intestine. Kraske (loc. cit.) cites Heuck² to the effect that in the Heidelberg clinic, in cases of high-seated rectal carcinoma in which, at

¹ *Journ. of Physiology*, 1913-14, xlvii, p. 57.

² "Zur statistische und operativen Behandlung der Mastdarm Krebse," *Verhandl. der deutschen Ges. f. Chir.*, 1883, xii, pt. 2, p. 199.

FIG. 1.



Showing a plaque of carcinomatous cells detached from one of the intact eminences of a tumour of the colon, by lightly rubbing a cover-glass over it; examined in glycerine. Low power magnification. A microscopic section of the growth after fixation confirmed its carcinomatous nature.

Explicatio figurae.

Epithelii cellularum lamina a coli carcinomate abrasa, superficie leniter tritendâ. Paulum magnificata.

operation, the lower, apparently sound, portions of mucosa had been left behind, recurrence not infrequently occurred in the latter. He himself concluded that the recurrences were due to the presence of implanted metastases which had taken place *before* the operation. It is, however, more probable that they arose from direct inoculation of the wound with free carcinoma cells within the lumen of the bowel.

Cell Infection of Wounds: Cytocides.

This brings us to the final subject of the present communication—viz., the consideration of what means are available for the disinfection or sterilisation of the surfaces of a cell-infected recent wound. The grosser forms of wound infection, such as those due to exploratory incisions into carcinomata and the pursuance of an operation with the same instruments, or to incisions thoughtlessly carried into peripheral parts of the growth during the progress of its removal; these are things of the past, for their danger has been long recognised. And the same is almost true of stitch cancer—the form, at least, which arises from the infection of the needle or suture by the contaminated fingers. Under some circumstances, nevertheless, it would seem that suture tracks through the abdominal parietes may be infected in a more insidious manner—viz., by the introduction of cells freed within the abdominal cavity from the infected peritoneum over a gastric or intestinal or a uterine carcinoma.¹ And Mr. Ryall recounts a case of abdominal hysterectomy for uterine carcinoma, where a limited infection of the abdominal wall took place, as shown by the appearance of a solitary cancerous nodule in the cicatrix some months after the operation, the recurrence being explicable after the same manner. Von Bergmann² refers to a case where after paracentesis of the abdomen (for what was thought to be tubercular peritonitis) the line of puncture became the seat of a carcinomatous infection. Similar cases of infection of the puncture made in tapping for cancerous ascites have been described by Courtin³ and Reinecke;⁴ and are stated by Guelliot (*loc. cit.*) to have been observed by Waldeyer, Nicaise, Quincke, and Terrillon. Under such circumstances the wound infection may be due to the

¹ Charles Ryall, *Brit. Med. Journ.*, 1908, ii, p. 1005.

² *Berl. klin. Wochenschr.*, 1887, p. 891.

³ *Assoc. française de Chirurgie*, 7 Congrès, 1893, p. 528.

⁴ *Arch. f. path. Anat.*, 1870, Bd. xxii, p. 391.

escape of free cancer cells with the ascitic fluid; or a nodular lesion of the parietal peritoneum may have been present, the perforation of which led to the liberation of cells and gave them access to the puncture. In the removal of carcinomatous organs, again, when the disease has perforated their capsules, the risk of wound infection is inevitable. In lateral pharyngectomy for carcinoma, where the diseased structures are reached through the side of the neck and have to be delivered through the wound, the same danger arises in the final stages of the operation.

Nor can we fail to perceive that the area around any carcinoma of a mucous membrane, seeing that it may be contaminated with shed and scattered cells, should be systematically sterilised prior to excision, as much against cells as against bacteria, and that the free surface of the neoplasm itself should be treated in every case in the same way, with the object of preventing the possible implantation of living cells on to the faces of the surgical wound.

What substances are available for such a purpose? Cytocides, they may be conveniently named. All bactericides are not cytocides, at least as tested from the standpoint of practical application, and some cytocides are not bactericides.

Before giving the detailed results of our experiments upon the action of inorganic solutions, we may recount certain observations of a less simple character which we made with the following objects—viz., to ascertain—

(1) Whether any cytolytic or cytocidal substance is produced by the liver during the growth of mouse carcinoma.

(2) Whether a selective anti-carcinomatous cytolysin can be raised by the heteroplastic grafting of mouse cancer into the rabbit.

(3) Whether any antagonism can be shown experimentally to subsist between tuberculosis and carcinoma—i.e., whether the toxic products of the bacillus are inimical to the cancer cell.

(1) ACTION OF THE LIVER OF MICE WITH GRAFTED CARCINOMA UPON MOUSE CANCER IN VITRO.

It was observed some while ago, at the Imperial Cancer Research, that the liver of mice which were growing spontaneous or grafted carcinoma underwent hypertrophy.¹ A direct proportion was found to

¹ F. Medigreccanu, *Proc. Roy. Soc.*, B, 1910, lxxxii, p. 286.

hold between the growth of the tumour and the weight of the liver. Dr. Bashford remarks,¹ in regard to this, that the hypertrophy is greater in animals bearing progressively growing tumours than it is in animals in which tumours are undergoing absorption; whence he inferred that the hypertrophy is concerned in promoting the growth of the neoplasm and not in hindering it. It was with the object of testing whether the hepatic overgrowth connoted the production of a specific cytocidal substance that the following experiment was carried out.

December 31, 1913: The pulp of two grafted tumours, of nineteen days' growth, was pooled and divided into two equal portions. To one portion an equal bulk of the finely minced liver of one of the mice from which the tumour had been removed was added and thoroughly mixed, after first allowing the blood of the minced liver to drain off. The mixture was incubated in a covered capsule at 37° C. for twenty minutes, and one division (0.05 c.c.) of a Bashford syringe was injected subcutaneously into each of six mice.

As a control experiment, an equal portion of the finely minced liver of a normal mouse was mixed with the other half of the tumour pulp and likewise incubated at 37° C. for twenty minutes. One division (0.05 c.c.) of a Bashford syringe was injected in the same manner into each of six mice.

January 9, 1914 (ten days): All the six mice injected with the tumour pulp, and liver from the carcinomatous mouse show growths at the site of inoculation, as likewise do all the six controls. The tumours in both sets average about the same size.

January 15 (sixteen days): Of the first set of six, three of the tumours are of good size, two are comparatively small, and one is no larger than a pea. Of the six controls, all but one had now grown tumours, the smallest of which was about the size of a pea.

The experiment was now closed.

Result: The liver of the mouse in which grafted carcinoma is growing subcutaneously has no cytocidal action upon mouse carcinoma of the same strain when mixed *in vitro* with the pulp of the tumour to be grafted.

¹ "International Congress of Medicine," Lond., 1913: Sub-section III (a), Chemical Pathology.

(2) THE ACTION OF THE BLOOD SERUM OF THE RABBIT PREPARED BY THE PREVIOUS INTRAPERITONEAL INJECTION OF MOUSE CARCINOMA UPON GRAFTED MOUSE CANCER.

In the following experiments the rabbit was prepared by means of eight intraperitoneal injections of mouse tumour pulp, the injections being made at about weekly intervals; the total number of tumours used was twenty. The rabbit serum was obtained fresh for each group of experiments by bleeding the animal from the auricular veins. In five sets of experiments the serum was injected subcutaneously into mice in which a grafted carcinoma of the same strain had commenced to grow. In one further set the serum was injected simultaneously with, but on the side opposite to, the grafted material; and in one other set the serum was used prophylactically.

It will be quite sufficient, seeing that no results of a positive kind ensued, to give only brief details of the experiments.

The Prepared Serum used curatively.

Experiment 1.

The rabbit used in all the following experiments was prepared by means of intraperitoneal injections made on March 6, 11, 15, 21, 28; April 3, 11, 19 (1912), eight in all. It was a vigorous male of seven months.

April 24, 1912: The rabbit was bled from the ear by puncture, after the part had been thoroughly washed with ether soap, followed by ether. After the blood had clotted the serum was centrifugalised in order completely to clear it of cells; 1 c.c. of the serum was injected intraperitoneally into each of eight mice in which there were growing grafted carcinomata (Tumour No. 63, Imperial Cancer Research). They were all dead within twenty-four hours. In some the liver was blotched with extravasations.

Control Observation upon Normal Mice.

April 30: Two normal mice received each, intraperitoneally, 1 c.c. of the prepared rabbit serum. They were dead within twenty-four hours.

Experiment 2.

As we found that 0·5 c.c. of the serum given subcutaneously was tolerated by normal mice, this dose was adopted in all the following experiments.

May 10, 1912: 0·5 c.c. of the prepared serum was injected subcutaneously into each of four mice in which tumour grafting had been carried out on May 2, with positive results; the growths were about the size of barley-corns. The injection in these, as in all the succeeding experiments, was made on the side opposite to the tumour.

May 15: The injection was repeated. Three of the four mice died within a few days afterwards, with diarrhœa.

May 20: The survivor received a third injection. This animal was killed on June 3. The tumour was the size of a large pea, and, on section, presented no macroscopic necrosis or cyst formation. At the site of the serum injections there were two ulcers of the skin and some subjacent suppuration.

Experiment 3.

May 15, 1912: 0·5 c.c. of the prepared serum was injected subcutaneously into each of three mice which had grafted tumours of fair size in progress.

May 20: Injection repeated.

May 28: The animals were killed. In each the tumour had now grown to a large size; on section it exhibited central necrosis with growing peripheral foci, but nothing of an unusual character.

Experiment 4.

May 30, 1912: 0·5 c.c. of the prepared serum was injected subcutaneously into each of six mice in which grafted tumours had commenced to grow, but were quite small.

June 3: Two dead; the tumours were the size of peas. 0·5 c.c. injection repeated on the survivors.

June 4: One dead; the growth consisted of a chain of small nodules; the liver showed many white necrotic foci; diarrhœa.

June 6: 0·5 c.c. injection repeated.

June 10: 0·5 c.c. injection repeated for the fourth time.

June 11: One dead; tumour of moderate size. It presented nothing

noteworthy to the naked eye on section; the liver was speckled with necrotic foci.

June 20: Remaining animal killed; tumour of large size; liver normal to the naked eye.

Experiment 5.

June 3, 1912: 0·5 c.c. of the prepared serum was injected subcutaneously into each of four mice in which grafted tumours were growing.

June 6: 0·5 c.c. injection repeated.

June 7: One dead; tumour on section showed normally growing periphery; liver speckled with necrotic foci.

June 10; 1 c.c. injection given.

June 11: One dead; tumour showed nothing noteworthy; liver exhibited no necrotic foci.

June 14: One died; the tumour showed, on macroscopic section, normal growing areas; liver speckled with necrosis; diarrhoea.

June 20: The only survivor dead; the tumour showed, on macroscopic section, normal growing areas; liver speckled with necrosis; diarrhoea.

The Prepared Serum used simultaneously with the Grafted Tumour, the Injection being made on the Side opposite to the latter.

July 3, 1912: 0·5 c.c. of the prepared serum was injected on the left side, immediately after the tumour pulp had been inserted beneath the skin on the right.

July 15; Four of the six mice used have developed well-pronounced tumours.

July 24: These four have now large tumours, and were killed; liver and other organs normal.

July 29: The remaining two, which had not grown tumours, were killed; liver and other organs normal.

The Prepared Serum used prophylactically.

June 24, 1912; 0·5 c.c. of the prepared serum, injected subcutaneously into each of seven mice.

July 1: Two dead; liver showed well-marked necrotic foci; diarrhoea.

July 3: On the opposite side an insertion of cancer pulp was made.

July 15: The five survivors have all developed tumours.

July 22: One dead; tumour large; no necrosis in liver.

July 24: The remaining four killed. All have tumours of full size; the liver of each was normal.

GENERAL SUMMARY AND CONCLUSIONS UPON THE FOREGOING EXPERIMENTS.

It appears from these experiments that the mouse-tumour-rabbit blood serum obtained from the rabbit after the eight times repeated intraperitoneal insertion of mouse cancer pulp has no specific cytolytic action upon growing grafted mouse cancer of the same strain, even though the injection of the serum may be several times repeated. The serum is not so specific but that it produces multiple focal necrosis in the liver, and if it brings about any death in the tumour this is not so general as to avail in effecting a curative retrogression.

Seeing that the particular mouse tumour used (No. 63, Imperial Cancer Research) normally undergoes central necrosis in the course of growth, it was not possible to determine whether this was increased or not in amount by the action of the serum. The mice from which the tumours were obtained were in all cases killed by instantaneous decapitation, and allowed to bleed in warm water; but, notwithstanding this, the tumours were not strictly bloodless; and the presence of mouse blood doubtless added to the cytolytic properties of the rabbit serum.

The action of hæmolytic sera on the tissues was fully described by one of us (L. S. D.) in the Pathological Section of the Royal Society of Medicine.¹ In this communication it was shown that hæmolytic sera, besides producing a lytic action upon the blood, brought about necrosis likewise of the liver and kidney. But as regards the tumour itself, if any necrosis was induced in it this was, as already stated, not distinguishable from the natural necrosis which regularly occurs during the growth of the particular tumour used. And the practical result clearly is that the action of the mouse cancer cells upon the rabbit does not lead to the production of a serum of practical utility in the treatment of mouse cancer. *Mutatis mutandis*, it may be inferred that the same would be true of the human disease.

¹ L. S. Dudgeon, P. N. Pantou, and E. Athole Ross, "The Action of Splenotoxic and Hæmolytic Sera on the Blood and Tissues," *Proc. Roy. Soc. Med.*, 1909, ii (Path. Sect.), p. 64.

As a prophylactic the serum is equally valueless. Nor did any more positive result ensue when the serum was injected simultaneously with, but on the side opposite to, the grafted material.

(3) TUBERCULOSIS AND CARCINOMA.

The view that an antagonism exists between tuberculosis and malignant disease originated with Rokitansky,¹ who remarks that tuberculosis is especially distinguished by its exclusive relation towards several morbid processes, an exclusion which obtains particularly between this disease and carcinoma. Rokitansky's theory of crases underlies his writings both upon tuberculosis and carcinoma, and is now only of interest as indicating what a reflective observer was able to formulate in regard to the pathogenesis of these diseases before the advent of bacteriological discovery, and before Virchow's application of the cell theory of Schleiden and Schwann to pathological processes. Strange as it now seems, Rokitansky held that the anatomical lesions of both diseases arose not by local cell proliferation, but from the effusion of an exudate which furnished a blastema, out of which the lesions originated. With respect to the same organ Rokitansky contrasts the incidence of tuberculosis and carcinoma, thus:—

<i>Frequent.</i>	<i>Rare.</i>
Lung tubercle.	Lung carcinoma.
Ovarian carcinoma.	Ovarian tubercle.
Salivary gland carcinoma.	Salivary gland tubercle.
Stomach carcinoma.	Stomach tubercle.
Œsophagus carcinoma.	Œsophagus tubercle.
Rectum carcinoma.	Rectum tubercle.
Ileum tubercle.	Ileum carcinoma.

Most of these differences, as will be obvious, admit of explanations which do not require the assumption of any proper antagonism.

Nevertheless, the co-existence of tuberculosis and carcinoma is not a common one.² The two diseases may be present simultaneously in

¹ "Pathological Anatomy," English translation, 1846, i, p. 312.

² Roger Williams ("Natural History of Cancer," p. 338) found the two diseases in *progress* together only twice in 136 cancer necropsies; similar analyses by others give like results. Lubarsch (*Archiv f. path. Anat.*, 1888) found that of 569 cancerous persons, 20·6 per cent. presented tuberculous lesions, old or recent; whereas of 5,967 non-cancerous persons, 42·7 per cent. were tuberculous; so, of 2,668 tuberculous patients, he found 4·4 per cent. were cancerous, whereas of 3,868 non-tuberculous cases, 11·7 per cent. were cancerous.

different organs, or they may co-exist in the same, either in different positions or at the same spot. So far as the College Museum is concerned, there is one record of the second association, in which metastatic carcinomata occurred in lungs which were tuberculous. The primary neoplasm was at the pylorus.

2415A: The pyloric portion of a stomach. For a distance of 7.5 cm. (3 in.) above the pylorus the gastric mucosa is the seat of a slightly raised growth, which has invaded the muscular coat and led to secondary disease of the adjacent lymphatic glands. The greater part of the surface is ulcerated. Histologically the growth is a columnar-celled carcinoma of atypical character, the cell columns being without lumen and composed of polyhedral cells, bounded on the deep aspect by groups of columnar elements. From a man, aged 62, who died in a very emaciated condition, with great distension of the stomach. The autopsy revealed chronic phthisis with cavitation at the apices of both lungs; there were many carcinomatous metastases beneath the pleura.

Besides this there is in the College Museum a specimen of what is, perhaps, the best known local intergrowth—viz., the superaddition of carcinoma upon cutaneous tuberculosis—the condition named lupus-cancer by Sir Jonathan Hutchinson.¹ Disregarding those cases where the carcinomatous lesion merely originates in the lupus cicatrix as of little special interest, in others it arises in the tubercular area, which may not even be ulcerated.² The earlier monograph of Dr. Bayha³ contains a collection of forty-two cases of this association, of which five were observed by himself. So far from the diseases being antagonistic under these circumstances, the rapid growth of the carcinoma and its high malignancy in regard to recurrence after removal are features insisted upon by all those who have studied the subject. The example of lupus-carcinoma in the College Museum is the following, and it illustrates both the rapidity and wide extension of the superadded carcinomatous lesion:—

1693A: A vertical section of a left elbow. From the integument there has grown an extensive squamous-celled carcinoma, the surface of which is in varying degrees disintegrated. The articular end of the ulna has been laid bare by the destruction of the suprajacent soft parts, and has been detached from the shaft by a deep extension of the disease

¹ Hutchinson's "Archives of Surgery," 1891, ii, p. 138.

² Crocker's "Atlas," plate 60.

³ Brüns's *Beiträge z. klin. Chir.*, Tübingen, 1888, iii, pp. 1-21.

which traverses the remains of the articular cavity and projects from its anterior aspect. From a woman, aged 69, who first noticed the growth three or four months before death. The patient stated that about thirty years ago she observed a swelling "like a bean" on the forearm; the skin subsequently became scaly and thick around, and the latter condition gradually extended downwards towards the hand, and upwards towards the elbow.

The hand with the lower end of the forearm of the same case is likewise preserved (4078). The ends of the digits have in different degrees suffered destruction from ulceration and necrosis; the terminal phalanx of the little finger has been lost. In the living condition the skin of the hand and of the lower end of the forearm was thickened, scaly, and mottled with dark brown patches, and was the seat of a certain number of flattened nodules and of superficial ulcers. Microscopic examination of the cutaneous nodules from the forearm reveals the presence of typical giant-celled systems in the corium; and sections of the skin over the terminal phalanx of the little finger (which was detached during life) present equally characteristic appearances. This case has been recorded by Dr. P. S. Abraham in the *Transactions of the Pathological Society*, vol. xlii, p. 309.

In addition to this, the College Museum contains the following specimen showing the supervention of carcinoma upon a verruca necrogenica: A finger with the head of the metacarpal bone. On the dorsal aspect of the metacarpo-phalangeal articulation there is a circular convex elevation, the surface of which is formed of disintegrated, brownish-yellow, horny epithelium. Microscopic examination shows the disease to be a squamous-celled carcinoma. The disease arose at the site of a tuberculous "post-mortem tubercle" or verruca necrogenica, which had existed for many years.

Hutchinson states (loc. cit.) that he had seen several cases of lupus in which malignant tumours had originated in other parts—e.g., in the breast, in association with lupus of the face; this being further evidence, of course, that tuberculosis and carcinoma are not mutually exclusive diseases.

Of the three examples which have fallen under our personal observation, one is a case of squamous-celled carcinoma of the cervical lymphatic glands secondary to disease of the tongue, where the integument over one of the glands is the seat of typical tuberculosis. The tubercular condition of the skin was discovered only on microscopic examination, the skin having been removed with the glands, as it was

thought to have become involved; there was no ulceration. The case was under the care of Sir Rickman Godlee, and the histological examination was made by Dr. C. E. Shattock.

In the second case, a carcinoma of the uterus was associated with tuberculosis of lymphatic glands. The specimen, which is in the Museum of St. Thomas's Hospital, is as follows: A vertical section of the pelvic organs of a child, aged $2\frac{1}{2}$. Growing from the uterus, and distending the vagina and elongating the urethra (with the posterior wall of which it is incorporated), there is a voluminous tumour into which a certain amount of hæmorrhage has occurred, and which microscopic examination shows to be a carcinoma arising from the endometrium. For three weeks before admission there had been a discharge of blood from the vagina; the bladder and rectum acted normally. There was no family history of carcinoma. The child's father was aged 28 and her mother 23, the child herself being the firstborn. Examination disclosed a palpable tumour in the hypogastrium, lying between the bladder and rectum. A catheter specimen of urine contained no blood. An exploratory laparotomy by Mr. Adams showed the condition present to be inoperable. The child died two months later. At the autopsy several enlarged glands were found in the mesentery; the lungs were broncho-pneumonic, and the lymphatic glands in the thorax and neck were likewise enlarged. Microscopic examination of these glands showed them to be tuberculous, without being the seats of metastatic tumours. There were no secondary growths in the other abdominal organs. The kidneys were somewhat hydronephrotic.

The third example belongs to the most interesting of the different possible combinations of the two diseases already cited—viz., that in which they occur intermingled at the same spot. The part affected was the testicle, and the condition is sufficiently rare to be worth illustrating. The organ was excised from a man, aged 42, admitted into St. Thomas's Hospital under Mr. C. A. Ballance in October, 1911. There was no history of venereal disease or of injury. In August, 1910, the testicle became swollen and painful; a week later it had become hard and was free from tenderness. The iliac glands, which were enlarged, were removed with the testis; these proved to be carcinomatous.

Roger Williams (*loc. cit.*, p. 339) gives the following list of the co-existence of the diseases in the same locality. In the stomach (Claude); in the uterus (Wallert, and Franqué); in the mamma (Moak, Kallenberger, Crawford, and Warthin); in the liver (Frerichs); in the

FIG. 2.



A microscopic section from a testicle in which the lesions of tuberculosis are distributed in a spheroidal-celled carcinomatous growth arising in the tubuli seminiferi. In the connective tissue supporting the neoplasm there are many isolated and confluent giant-celled systems. ($\frac{2}{3}$ obj.)

Explicatio figurae.

Testiculi sectio in quâ laesiones ex tuberculose per tumorem malignum (carcinoma) sparguntur. Telae connexivae tumoris in septis monstrantur cellularum foci qui tuberculosis proprii sunt. Sectio paulum magnificatur.

large intestine (Dalton, Zenker, Naegeli, and Moak); in the rectum (Naegeli, and Baumgarten); in the small intestine (Lubarsch, and Naegeli); in the larynx (Baumgarten, Zenker, and Crone); in the lung (Batty Shaw, Schwalbe, Wolf, Wilson Fox, and Friedländer); in the œsophagus (Cordua); in the eye-sarcoma (Silex).

The most recent brochure upon the subject is that by Horst Oertel,¹ whose conclusions, drawn from a digest of recorded examples collected from different authors, are as follows: "It is plain . . . that neither tuberculosis nor cancer seem to possess specific antagonistic qualities, but that the results of such combinations depend upon the conditions of individual cases." "Thus it appears probable that a . . . rapidly growing cancer may overcome the resistance imposed upon it by an approaching or even freshly implanted tuberculous inflammation. On the other hand, an actively progressing virulent tuberculous infection may overgrow an approaching or superadded cancer. . . . Finally, both lesions may co-exist and intergrow without any marked influence upon one another, . . . unless by caseation the tuberculous infection removes the necessary conditions for any growth."

As every natural hint, however slender, in the direction of therapeutics is worth following up, we undertook two years ago a series of experiments with mouse cancer to ascertain whether the progress of the tumour could be arrested by tubercular infection, in the hope that if this were so, tuberculin might be of service as an agent in the treatment of carcinoma. Nothing, however, of any practical applicability came from the experiments, which may, notwithstanding, be worth briefly recording.

Tubercle Bacilli, Living, used curatively as a Subcutaneous Injection.

May 10, 1912: Six mice, which had been inoculated eight days previously with mouse cancer, and in each of which a small growth was present, received 0.5 c.c. of a salt suspension of a culture of tubercle bacillus raised from a human source on Dorset egg medium, the injection being made on the side opposite the tumour.

May 15: 0.5 c.c. injection repeated.

May 25: Four of the animals have now large tumours; and the other two, tumours of smaller size.

¹ *Journ. of Med. Research*, Boston, 1911-12, xxv, pp. 503-514.

Tubercle Bacilli, Dead, used curatively as an Intratumoral Injection, followed by the Subcutaneous Injection of Tuberculin.

January 9, 1912: Eight mice, with growing grafted tumours of seventeen days' age. Into the substance of each tumour was injected a thick suspension of dead tubercle bacilli (as supplied by the firm of Lucius) suspended in salt solution.

January 17: Each animal received 0.25 c.c. of a strong solution of glycerine-free tuberculin in salt solution, the injection being made on the side opposite the tumour.

January 27: Six killed. The tumours had grown to a full size. On macroscopic section they exhibited nothing noteworthy; there was no suppuration; though centrally necrosed (as normally happens in the case of this particular strain) they presented translucent growing areas at the periphery. Of the other two mice, one developed a large tumour, and in the other the original growth retrogressed.

Tubercle Bacilli, Living, mixed with the Cancer Pulp, the Mixture being injected subcutaneously.

November 20, 1912: The pulp of three tumours of fourteen days' growth was pooled, and divided into two parts, with one of which was mixed a small quantity (five or six drops) of a suspension in salt solution of a vigorous culture of tubercle bacilli, raised from a human source on Dorset egg medium. Five mice were subcutaneously injected with the mixture. To a second part of the pulp considerably more of the living tubercle bacilli were added; with the mixture seven mice were injected.

December 12 (twenty-two days): Of the first group, three have grown tumours, two have not. Of the second group, five have grown tumours, two are negative. The mice were now killed and the several tumours divided for macroscopic inspection. They presented no unusual appearances.

Tubercle Bacilli, Living, mixed with the Cancer Pulp, the Mixture being injected subcutaneously; followed by the Subcutaneous Injection of Koch's Old Tuberculin.

April 3, 1913: Two tumours of eighteen days' growth were pooled, and the pulp thoroughly mixed with a vigorous culture of tubercle bacilli raised from a human source on Dorset egg medium. Four mice were injected subcutaneously with the mixture.

April 18 (fifteen days): Only one animal developed a tumour of a size worth testing. This received 1 c.c. of 1:1,000 Koch's old tuberculin subcutaneously on the side opposite to the tumour. The injection was repeated on April 21, 24, and 28, on the back; and, on May 1, $\frac{1}{10}$ c.c. of Koch's old tuberculin was injected without dilution.

May 8: Killed. The tumour was large; on macroscopic section it presented no notable vascularity, and no suppuration had occurred within it; there were no lesions in the lymphatic glands or internal organs. None of the injections produced any general illness.

Tubercle Bacilli, Living, injected subcutaneously as a Prophylactic.

January 19, 1912: Five mice received subcutaneously on the left side 0.5 c.c. of a culture of tubercle bacillus raised from a human source on Dorset egg medium. This was repeated on January 30 and February 6.

February 14: The animals were grafted on the right side with mouse tumour pulp.

March 9: Four of the mice have developed tumours, which subsequently continued to grow, although their development was at first retarded.

Tubercle Bacilli, Dead, injected subcutaneously as a Prophylactic.

January 9, 1912: Seventeen mice were injected subcutaneously with a thick suspension in salt solution of dead tubercle bacilli (as supplied by the firm of Lucius).

January 17: Each was grafted on the opposite side with tumour pulp. Tumours subsequently grew in thirteen of the animals, without any pronounced retardation or reduction in size.

THE ACTION OF INORGANIC AND OTHER SOLUTIONS AS CYTOCIDES.

The method adopted in order to test the cytocidal value of inorganic or other substances in solution was to submit the tumour pulp to their action for a measured time in a hollow block of glass, and then to graft the material so treated, subcutaneously, on a series of mice, and observe whether the insertion was followed by the growth of a tumour.

The tumours used, like all those in the foregoing experiments, were raised from that known at the Imperial Cancer Research as No. 63,

the original of which was a spontaneous carcinoma of the mouse's mamma, and which is now so amenable to grafting that, of ten mice inoculated, eight or nine, and not rarely all, will grow tumours. The tumours used were of about fourteen days' growth. The technique adopted was as follows: The mouse was etherised, and, when insensible, was nearly decapitated from the dorsal aspect, and placed in a basin of warm water, into which the blood was pumped out by the action of the heart; the object of this was to obtain the tumour as free of blood as possible. The growth was then removed in the usual way by peeling off the skin and lifting it from its bed; it was then transferred to a hollow block of glass or "capsule," and picked to pieces with two pairs of forceps so as to remove fascia and all but the finest connective tissue, and leave a uniform, diffuent pulp of cells. The solution to be tested was then poured on in quantity, and with curved scissors the pulp was distributed, and still further subdivided during the whole time the solution was allowed to act. The fluid was then pipetted off, and the material injected subcutaneously with a Bashford syringe.

In most cases the pulp, before injection, was washed in distilled water, which was finally pipetted off. The object of this was partly to bring the action of the solution to an end and partly to obviate the occurrence of any necrosis of the delicate skin or abdominal muscles at the site of the inserted material, which might lead to the extrusion of the latter and so invalidate the result, a thing which happened less than half a dozen times in the whole of the experiments. Needless to say, everything was carried out with strict aseptic details.

We may place first, the series of observations of which the result was negative, and conclude with those in which the solutions proved to be efficient cytocides.

Distilled Water.

The simplest of all cytocides or agents lethal to isolated mammalian cells is distilled water, which kills, of course, by the endosmosis it induces. Upon the finely divided carcinoma pulp it is inefficient, doubtless because the cells are not sufficiently isolated to come individually under its action. For practical purposes, therefore, it would be useless, as the following experiment will show:—

April 30, 1914: The water used was specially prepared the day previously in all-glass apparatus. The capsule was flooded, and, after a while the material was allowed to settle, the slightly blood-stained

fluid being pipetted off and the water renewed. After fifteen minutes in all, the water was removed, and one division (0.05 c.c.) of a Bashford syringe given subcutaneously. The material swelled up and became sticky and pale during its preparation; nine mice were injected.

May 14 (fifteen days): Six show tumours ranging from the size of a pea to that of a haricot; three are negative.

May 21 (twenty-two days): Two of the tumours are large, four of medium size. Three mice show no growth. The experiment was now closed.

Result: Distilled water, allowed to act fifteen minutes, is valueless as a cytocide from the practical standpoint; six tumours appearing in nine mice inoculated.

Colloidal Mercury.

In the next experiments we made trial of colloidal mercury and colloidal selenium, both of which are potent bactericides. Colloidal mercury (H. Crookes) is a suspension of 1 in 2,000 of metallic mercury in a filter-passing state, but visible beneath the ultra-microscope. *Bacillus coli* is killed by it in ten seconds, staphylococci and streptococci in from three to four minutes.¹

April 28, 1914: The tumour pulp was flooded with a freshly prepared suspension of colloidal mercury for three minutes, the material being kept distributed and still further subdivided by means of curved scissors during the whole of the time. By an independent observation we ascertained that, when the blades of the scissors were kept submerged in the colloid for fifteen minutes, no visible deposition of mercury took place. The fluid was then removed by throwing the material into a porcelain sieve; the pulp had become swollen like boiled sago. It was not washed afterwards with distilled water. Before the first injection was made, seven minutes had passed from the commencement of the experiment. Nine mice were inoculated. The animals were individually examined on April 30, May 1 and 5, to see whether any sloughing of the overlying skin had occurred at the site of injection; the local condition was found to be perfect.

May 12 (14 days): Of the nine mice, seven have grown tumours, the smallest of which was the size of a pea; the others were all well declared. The experiment was now closed.

¹ *Brit. Med. Journ.*, 1912, i, p. 521.

Result: Colloid mercury, allowed to act three minutes, is valueless as a practical cytocide; seven out of nine mice inoculated, growing tumours.

Here we may add that, in a series of experiments made previously to those just given, with colloidal mercury and colloidal silver, when these were first introduced by H. Crookes, we found that both were powerless to stop the growth of grafted mouse cancer, whether injected interstitially into the tumour whilst as yet small, or used subcutaneously on the side opposite the growing neoplasm.

Colloidal Silver (as a Subcutaneous Injection).

March 5, 1912: Four mice, with grafted tumours of nineteen days' growth, received each 0.5 c.c. of colloid silver subcutaneously on the side opposite to that on which the tumour was growing. The injection was repeated on March 8, 11, and 13. The animals were killed on March 16. The tumours had grown to a large size, and, on being cut through, exhibited nothing unusual to the naked eye.

Colloidal Mercury (as a Subcutaneous Injection).

January 25, 1912: Two mice, with grafted tumours of fourteen days' growth, received each 0.5 c.c. subcutaneously on the side opposite that on which the tumour was growing. The injection was repeated on January 29 and 30. The animals were killed on February 7, by which date the tumour in each had grown to a large size. The macroscopic appearances of the growths on section were of the usual kind.¹

Colloidal Selenium.

The preparation used was that made by the electrical method, from the clinical laboratories of Comer and Co., Paris. This suspension has been used intravenously in the treatment of malignant tumours in man, and is free from the toxic properties of Wassermann's eosin-selenium, which we have been unable to obtain.

July 13, 1914: The pulp of three tumours was flooded in a capsule

¹ In the *Journ. of Exper. Med.*, November 1, 1914, M. S. Fleisher and L. Loeb describe a retarding effect produced upon the growth of mouse cancer by colloidal copper, platinum, gold; the solutions were injected intravenously, the caudal veins being used; but in some cases the injection was made subcutaneously.

with colloid selenium for five minutes, during which time the material was kept distributed, and still further subdivided with curved scissors. The fluid was then pipetted off and all-glass distilled water substituted. The pulp did not whiten or coagulate. Ten mice were injected subcutaneously, each receiving one division (0.05 c.c.) of a Bashford syringe. The animals were examined individually on July 16 and 18, the site of puncture and the skin over the inserted material being found perfectly normal.

July 27 (fourteen days): Nine of the ten mice have well-declared tumours. The experiment was now closed.

Result: Colloid selenium, allowed to act five minutes, is valueless as a practical cytocide, nine of ten mice inoculated, growing tumours.

Peroxide of Hydrogen.

October 7, 1914: The pulp of four tumours, of three weeks' growth, was placed in a small bag extemporised from four folds of sterilised gauze of open texture, and, the mouth being held in a pair of forceps, the whole was submerged in a small beaker of peroxide of hydrogen (10 vols., British Pharmacopœia), and kept briskly moving for five minutes, the peroxide being once changed during the process. The bag was then washed in all-glass distilled water, and the pulp removed from it, and used to inject nine mice, each receiving one division (0.05 c.c.) of a Bashford syringe.

October 10: The animals were examined individually; the general and local conditions were perfect.

October 16: All were growing tumours.

October 21 (fourteen days): All have well-pronounced tumours.

Result: Peroxide of hydrogen (10 vols., British Pharmacopœia), allowed to act five minutes, the material being afterwards washed in distilled water, has no effect in inhibiting growth; of nine mice inoculated, every one grew a tumour.

Radium Emanation.

October 2, 1914: The pulp of four tumours, of sixteen days' growth, was flooded in a capsule with radio-active water for fifteen minutes, during the whole of which time the material was kept distributed, and still further subdivided with curved scissors; the fluid was partly pipetted off and replaced once during the process. After finally

removing the fluid, the pulp, without being washed in distilled water, was inserted subcutaneously into nine mice in the usual manner.

The experiment was started at 2.36 p.m., but as the removal of the fluid was not easy, since the material did not readily subside, it was 3 o'clock before the first injection was made. The radium emanation, which was quite freshly, and specially prepared, and stored in a stoppered bottle without an air space, was supplied to us from the Radium Institute by the courtesy of Mr. Pinch and Mr. Hogley. The contact of the scissors used in the technique would be quite innocuous to its potency.

It may be here pointed out that, although the alpha rays are particularly powerful, they possess little penetration in comparison with the beta and gamma, and in practice have little chance of acting upon tissue in bulk. This drawback, although it would be largely obviated in the conditions of the experiment, would, however, not be altogether removed; for it must be borne in mind that the greater number of the carcinoma cells are not individually isolated, but aggregated in minute clusters.

October 5 and 10: The general condition was perfect. Examination of the animals individually showed that the puncture and area over the inserted material were in every way normal.

October 16 (fourteen days): Of the nine mice, seven have grown tumours of varying sizes. The tumours continued to progress and the animals were subsequently killed at different dates to furnish material for other experiments.

Result: Radium emanation, allowed to act fifteen minutes (the material not being afterwards washed in distilled water), has no effect in inhibiting growth, seven out of nine mice inoculated growing tumours.

Taurocholate of Soda, 2 : 100.

November 16, 1914: The pulp of tumours of eighteen days' growth was flooded for ten minutes with a 2 per cent. solution of Merck's taurocholate of soda in distilled water, the fluid being once changed; the solution was then pipetted off and the material twice washed in all-glass distilled water. The pulp became curiously swollen and adhesive. Nine mice received each 1 division (0.05 c.c.) of a Bashford syringe subcutaneously, a distinct swelling being produced at the site of insertion. The solution itself is neutral.

November 20: The animals were individually examined, the

puncture and skin over the inserted material being found absolutely normal. The general condition had at no time been affected.

November 25: The same report; in none is there any sign of tumour growth.

November 30 (fourteen days): The animals individually examined. In four of the nine there is a growth about the size of a pea. The puncture and area over the inserted material is quite normal; the general condition perfect.

December 5 (nineteen days): The animals individually examined. In seven there are now growths of varying dimensions; the largest, the size of a haricot; the smallest, that of a pea. The experiment was now closed.

Result: Taurocholate of soda 2 per cent. in distilled water, allowed to act ten minutes, the material being afterwards washed in distilled water, delayed the appearance of the tumours, but without inhibiting their growth, which took place in seven out of nine mice inoculated.

Mercuric Perchloride 1 : 1,000.

We made trial of this as a cytocide in varying strengths. In a solution of 1 : 1,000 it is valueless, as the following experiment will show:—

May 25, 1914: Tumour pulp was flooded in a capsule for two minutes in 1 : 1,000 solution of corrosive sublimate in distilled water, with the details given in the preceding experiments. We ascertained by an independent observation that no visible deposition of mercury took place upon the blades of the curved scissors used, when submerged for fifteen minutes in the solution. The solution was pipetted off, but the material was not afterwards washed in this case with distilled water. The animals were individually examined on May 27 and 29, and June 4, the site of puncture and that of the grafted material being found normal.

June 8 (fourteen days): Of the ten mice seven have fully declared tumours, the other three show none. The experiment was now closed.

Result: Mercuric perchloride 1 : 1,000, allowed to act two minutes, is valueless as a practical cytocide, seven out of ten mice inoculated growing tumours.

The foregoing solutions were inefficient as cytocides, and we may conclude by detailing those which proved effectual, and which might be, therefore, of utility in surgical practice.

Mercuric Perchloride 1 : 500.

June 19, 1914: The tumour pulp was flooded in a capsule with a 1 : 500 solution of corrosive sublimate for *two minutes*, &c. The fluid was then pipetted off, and all-glass distilled water substituted; the latter was similarly removed, and a second amount added; this was pipetted off, and the material injected subcutaneously, one division (0.05 c.c.) of a Bashford syringe being given to each of twelve mice. We ascertained by an independent observation that no deposit of mercury took place on the blades of the scissors after five minutes' submersion; after fifteen minutes, however, there was an obvious deposition. Under the action of the mercuric solution the material became white and opaque from coagulation. The animals were examined individually on June 21 and 29; the site of puncture and skin over the grafted material remained normal.

July 7 (eighteen days): Two of the twelve mice show a growth, the others are negative.

July 14 (twenty-five days): The tumours had doubled their size, each was somewhat larger than a haricot. No trace of growth was present at any time in the other ten. The experiment was now closed.

Result: Mercuric perchloride 1 : 500, allowed to act for two minutes, the material being afterwards washed in distilled water, though giving a good result, did not prevent growth occurring in two of twelve mice inoculated.

Mercuric Perchloride 1 : 500.

July 14, 1914: The tumour pulp was flooded in a capsule with a 1 : 500 solution of corrosive sublimate for *five minutes*, &c. The fluid was pipetted off, and the material twice washed in all-glass distilled water; the mercuric solution rendered it white and opaque, and somewhat difficult to take up into the syringe. Eight mice were injected subcutaneously, a distinct local swelling being visible at the grafted spot.

July 17: Three of the animals were dead. Post mortem: Nothing abnormal at the site of injection. Of those alive, the site of operation on them was quite normal—i.e., both the puncture and the skin over the inserted material.

July 21: One dead. Post mortem: Site of operation in a perfect condition.

July 27 (fourteen days): In the four surviving mice no tumours have grown.

August 4 (twenty-two days) : The animals were killed. On reflecting the skin no trace of the grafted material was discernible, and there was no scar over the spot, showing that no necrosis of the integument had taken place.

Result : The death of four of the eight mice must be attributed to mercurial poisoning, the mercury being slowly absorbed from the albuminate formed in the inserted material. It is to be noted that all the animals were off food for some days, and became thin in a way not observed in other cases. The survivors regained good condition before the conclusion of the experiment. Mercuric perchloride 1 : 500, allowed to act *five minutes*, the material being afterwards washed in distilled water, prevented growth in four out of eight mice, four dying during the first seven days from mercurial poisoning.

Mercuric Perchloride 1 : 500.

July 28, 1914 : The tumour pulp was flooded in a capsule with 1 : 500 solution of corrosive sublimate for *five minutes*, &c. The fluid was partly pipetted off after three minutes, and a further amount of the solution added. After five minutes in all, the fluid was pipetted off and the pulp was twice washed with all-glass distilled water. Ten mice were injected subcutaneously, a visible swelling being produced in each at the site of insertion. As in the two preceding experiments, the material became white and opaque from coagulation.

July 31 : One was dead. Post mortem, on reflecting the skin the site of injection was found in a perfect condition, the grafted material being in good amount ; the skin and abdominal muscles in contact with the material were quite intact ; the subcutaneous fascia in contact with it was whitened. None of the animals have taken food well. One was moribund and was killed. Post mortem : Site of grafted material in a perfect condition ; the tissue in immediate contact with the latter was whitened, but the cutis was intact and so was the peritoneum. The whitening around the graft is, of course, due to the action of sublimate transuded from the treated material.

August 1 : A third dead ; post mortem, the same findings.

August 2 : A fourth dead ; post mortem the same findings. All the animals take little food.

August 4 : Two more dead ; post mortem, site of injection in perfect condition.

August 12 (fifteen days) : The four surviving mice are thin, but lively ; no tumour has grown in any.

August 21 (twenty-four days): These four were killed; (a) site and organs normal; (b) site and organs normal; (c) site and organs normal; (d) site, normal; organs, pale; kidneys, pale, surface finely speckled with uniformly and closely distributed white points. Microscopic examination of the kidneys showed the calcareous depositions in many of the convoluted and other tubules, which have been found in the human subject in cases of sublimate poisoning. Treatment with Sudan III revealed no fatty change in the renal epithelium. In the final stage of calcification the tubuli (in the mouse's kidney) are completely filled, as far as the basement membrane, with solid cylinders of calcium salts; in the earlier stages discrete blocks result from the calcification of individual cells of the necrotic or damaged epithelium. The material readily dissolves in dilute hydrochloric acid.

Result: Mercuric perchloride 1:500, allowed to act five minutes, the material being afterwards washed in distilled water, prevented growth in four out of ten mice, six dying during the first seven days from mercurial poisoning.

Mercuric Perchloride 1:600.

August 20, 1914: The tumour pulp was flooded with 1:600 mercuric perchloride for five minutes, &c., the fluid pipetted off, the material washed three times with all-glass distilled water, and (the water being finally removed with the pipette) used to inject ten mice subcutaneously, a local swelling appearing at the site of insertion in each.

August 24: Animals in good condition and feed well. On individual examination the site of puncture and the skin over the inserted material were quite normal.

August 26: One dead. Post mortem: On reflecting the skin no trace of the puncture was discoverable; the skin over the inserted material was normal; there was no whitening of the tissue in contact with the latter, which appeared as a small localised flattened oval mass. The lungs were almost solid from acute pneumonia. The other nine are in excellent condition, and examined individually present no trace of any local abnormality.

August 29: The same report.

September 3: No tumour has grown in any.

September 10 (twenty-one days): The nine mice killed. They are all in good condition. On reflecting the skin no trace of the puncture was discoverable in any; the skin over the site of the inserted material

was quite normal, without trace of scar or hairless area indicative of a past necrosis. The organs were all normal.

Result: Mercuric perchloride 1:600, allowed to act five minutes, the material being afterwards washed in distilled water, prevented growth in nine of the ten mice experimented with, the tenth dying of acute pneumonia six days after the insertion. The animals remained throughout in excellent general condition. We may lay stress on the last fact, since it is well recognised that the growth of mouse cancer is markedly delayed or even prevented when the state of general nutrition is much reduced. And not only is this true of mouse cancer, but the same observation has been made in the human subject, and was the basis of the starving method of treating the disease, carried out in past times with some degree of success by Hufeland.

Iodine, 1:100 Aqueous Solution.

August 5, 1914: Tumour pulp flooded in a capsule with 1 per cent. aqueous solution of iodine (with iodide of potassium); allowed to act *four minutes*, &c. The solution was pipetted off and the deeply brown-stained material was washed three times in all-glass distilled water. The water was finally pipetted off and the material injected subcutaneously into seven mice, a distinct swelling being produced in each at the site of insertion. A microscopic examination of the iodine-treated and washed material, in dilute glycerine showed the cells composing the groups to be all stained yellow, without further recognisable change.

August 29 (twenty-four days): The animals were killed and examined. In five, on reflecting the skin, a trace of brown debris was exposed at the site of the inserted material; the overlying integument was quite intact and without any trace of scar. In two, on peeling off the skin, a small scar was torn open over the site of the inserted material, showing that the delicate integument had undergone necrosis from the action of the iodine, and in these cases nullified the result. The general condition of the animals had never suffered.

Result: Aqueous solution of iodine, 1 per cent., allowed to act four minutes, the material being afterwards washed in distilled water, is an efficient cytocide.

Iodine, 1 : 100 Aqueous Solution.

October 29, 1914: The tumour pulp was flooded in a capsule with 1 per cent. aqueous solution of iodine (with iodide of potassium), allowed to act *eight minutes*, the solution being partly pipetted off and replaced, &c. The fluid was removed and the deeply brown stained material was twice washed with all-glass distilled water. The water was finally withdrawn and the material injected subcutaneously into eleven mice, enough to produce a distinct local swelling—between a half and one division (0.05 c.c.) of a Bashford syringe. The animals were examined individually on November 3 and 7; the site of puncture and the area over the inserted material were perfectly normal. The general condition had never been affected.

November 12 (fourteen days): In none has any tumour grown. The puncture and the area over the inserted material are in perfect condition, and the animals quite well.

November 19 (twenty-one days): No growth had occurred in any. All were then killed, and an autopsy was made upon each. On peeling off the skin the site of puncture and the skin over the insertion were perfectly normal; there was no trace of the grafted material.

Result: Aqueous solution of iodine in iodide of potassium, allowed to act eight minutes, the material being afterwards washed in distilled water, prevented growth in the eleven mice experimented with. The animals remained throughout in excellent general condition. The solution is thus an efficient cytocide.

Iodine, 2 : 100 Aqueous Solution.

August 20, 1914: The tumour pulp was flooded with 2 per cent. aqueous solution of iodine (with an equal part of potassium iodide—the minimum that will effect its solution) for five minutes, &c.; the fluid pipetted off, and the deeply brown stained material washed three times with all-glass distilled water, the water being finally removed with the pipette. Nine mice were injected subcutaneously, a local swelling being visible in each at the site of insertion.

The animals were examined individually on August 24 and 26, when the site of puncture and the skin over the inserted material were found to be in a normal condition. The general health had not at any time suffered.

September 3 (fourteen days): No tumour has appeared in any of the

animals. In two of them there is a small scar at the side of the inserted material.

September 11 (twenty-two days) : The nine mice were killed. Post mortem : In seven, on reflecting the skin, no trace of abnormality was to be seen either at the puncture or over the site of the inserted material ; viscera normal ; animals well nourished. In two there was a small scar over the site of the graft, which could be torn open. No growth had occurred in any of the nine.

Result : Aqueous solution of iodine, 2 per cent., allowed to act five minutes, the material being afterwards washed in distilled water, prevented growth in seven of nine mice ; in the remaining two a small scar was found over the site of the inserted material, indicating that the iodine had produced necrosis of the skin, which in the mouse is extremely thin and delicate. The solution is thus an efficient cytocide.

Iodide of Potassium, 2 : 100 Aqueous Solution.

This solution was used as a check to those of iodine, since the latter cannot be made without the addition of this salt ; it was prepared with all-glass distilled water.

October 29, 1914 : The tumour pulp was flooded in a capsule with the solution, which was allowed to act five minutes, and was once partly changed, &c. The fluid was pipetted off, and the material twice washed with all-glass distilled water. The water was finally removed, and the pulp injected subcutaneously into seven mice, in each enough to produce a distinct local swelling.

The animals were examined individually on November 3 and 7 ; the site of the puncture and the area over the inserted material were perfectly normal. The general condition had never been affected.

November 12 (fourteen days) : In six of the seven a tumour has grown ; in each case it is about the size of a small haricot.

Result : Aqueous solution of iodide of potassium, 2 per cent., allowed to act for five minutes, the material being afterwards washed in distilled water, has no effect in inhibiting growth, which occurred in six out of seven mice inoculated.

Iodine : Tinctura Iodi (British Pharmacopœia), 60 minims to 20 fluid ounces of Distilled Water.

December 18, 1914 : The tumour pulp was flooded with the above solution for ten minutes, the fluid being twice renewed ; the colour of

the latter was discharged on the first two occasions. The pulp was finally washed in all-glass distilled water. Eight mice received, each, 0.05 c.c. subcutaneously, a visible swelling being produced.

December 21: The animals were individually examined; the puncture and skin over the inserted material were in a perfect condition.

December 28 (eleven days): An examination of the animals individually showed nothing abnormal; no growth was apparent at the site of insertion.

January 7, 1915 (twenty-one days): In five of the eight mice tumours are now declared, though in none are they of the size they should be after three weeks, the treatment having evidently retarded growth; the tumours had all steadily, though very slowly, increased since the fourteenth day, when they were hardly more than indicated.

Result: Tincture of iodine (60 minims to 20 fluid ounces of water), allowed to act for ten minutes, the material being afterwards washed in distilled water, is unreliable as a cytocide, although experience shows it to be an efficient irrigant in bacteria-infected wounds. Growth took place in five of eight mice inoculated.

Sulphate of Zinc 1 : 240.

November 21, 1914: The tumour pulp was flooded for nine minutes with a solution of sulphate of zinc, made with all-glass distilled water, 2 gr. to the ounce—i.e., 1 : 240. The solution was once changed, then pipetted off, and the material twice washed with all-glass distilled water; it became opaque and difficult to manipulate. Six mice received each subcutaneously 0.05 c.c. of the material, a distinct local swelling being produced.

November 25: The animals were individually examined. The puncture and skin over the site of insertion were perfectly normal. The general condition had not been at all affected.

November 30 (nine days): No growth has appeared in any of the animals; the puncture and skin over the site of insertion are quite normal, the general condition perfect.

December 5 (fourteen days): No trace of any growth has appeared; the puncture and skin over the site of insertion are perfectly intact.

December 12 (twenty-one days): There was no trace of growth in any of the animals; they were now killed. On reflecting the skin no indication of any scar was detectable over the site of the inserted material, nor any remnants of the latter.

Result: Aqueous solution of sulphate of zinc 1 : 240, allowed to act nine minutes, the material being afterwards washed in distilled water, prevented growth in all of the six mice inoculated.

Absolute Alcohol.

December 11, 1914: The tumour pulp was flooded for eight minutes with absolute alcohol, the fluid being once renewed; after pipetting off the alcohol, the pulp, which had become opaque and somewhat white from coagulation, was washed in all-glass distilled water. Ten mice received each subcutaneously 0.05 c.c. of the material, a distinct local swelling being produced.

December 15: The animals were individually examined. The puncture and the skin over the inserted material were perfectly normal.

December 21 to 28: Same report.

January 1, 1915 (twenty-one days): In none of the ten mice has any tumour appeared.

Result: Absolute alcohol, allowed to act eight minutes, the material being afterwards washed in distilled water, prevented growth in all the ten mice inoculated.

Pathological Section.

May 4, 1915.

Dr. F. W. ANDREWES, President of the Section, in the Chair.

The Importance of Tyrosine as an Aid in the Demonstration of the Present-day *de novo* Origin of Living Organisms.

By H. CHARLTON BASTIAN, M.D., F.R.S.

THE incredulity that has been expressed by so many concerning the results of experiments destined to throw light upon the question of the present-day occurrence of so-called "spontaneous generation"—the process which I term "Archebiosis"—must certainly be regarded as testifying to the great importance that would be attached to a positive solution of this much contested problem.

This incredulity has given rise to attempts to explain away the results by one or other of the three following suppositions:—

(1) The bodies that are found are not organisms, but mere simulacra or pseudo-organisms.

(2) If real organisms have been found, others say, they are not living but dead organisms which pre-existed in the experimental solutions and have been killed by the sterilising process.

(3) The alleged organisms have not really been taken from the tubes, others surmise, but have come from an impure pipette, or have dropped from the atmosphere on to the microscope slip before the cover-glass has been applied.

These last suppositions are surely unworthy of serious consideration as attempts to explain the results of a prolonged research involving the preparation and examination of the contents of over a thousand experimental tubes containing different kinds of solutions, some of which have yielded negative and others positive results.

In the first place, the pipette is always carefully sterilised in the flame of a spirit lamp or of a Bunsen's burner before it is introduced into the just-opened tube in order to withdraw a sample of the deposit which it contains. Then, again, in trials with different solutions six or more tubes of the same kind have been similarly treated, and if organisms are found in the deposit of the first one examined the others of the same series have almost always been found to contain similar organisms;

while if, in another series, no organisms are found, others of this same series will be similarly barren. Moreover, when organisms are found, although some are very minute they may be very numerous in the midst of the deposit, either singly or in groups, and it would be quite absurd to suppose that they, or minute moulds, had all dropped from the

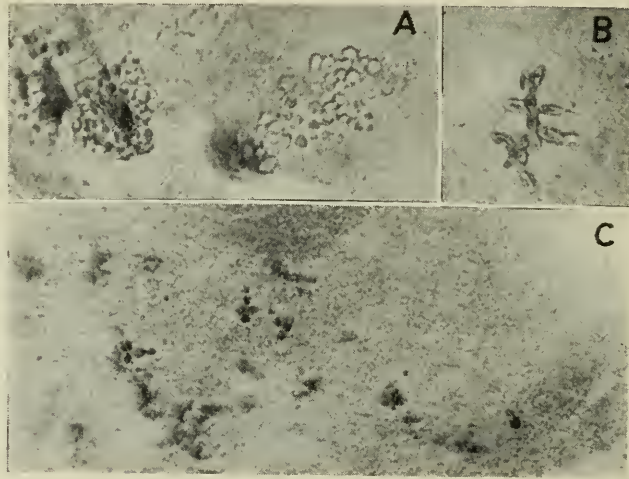


FIG. 1.

A, two groups of elementary organisms ($\times 500$); B, a germ developing ($\times 500$);
C, torulae (stained) in the midst of granular deposit ($\times 300$ only).

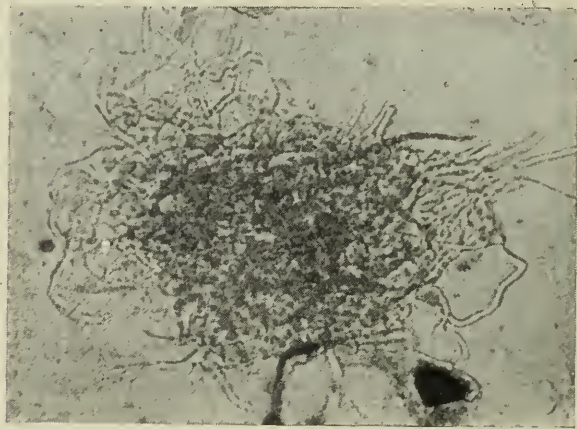


FIG. 2.

A bunch of *Leptothrix* filaments, slightly stained ($\times 500$).

atmosphere on to the slide during the few seconds before the application of the cover-glass. Further, if the atmosphere is to be the source, how would those who suggest it explain the non-appearance of organisms when the deposits of barren series of tubes are being examined? A few specimens of organisms as taken directly from the tubes before the addition of tyrosine are represented in figs. 1 to 3.

The other two lines of criticism are worthy of more serious consideration. In regard to (1), it is perfectly true that pseudo-organisms most closely resembling bacteria of different kinds or minute cellular organisms are not infrequently to be met with in the deposits from the experimental solutions; but so also, and much more frequently, are real organisms. And in regard to (2), if organisms pre-existed in the experimental fluids, as they not infrequently do, they would undoubtedly be killed by the three boilings for twenty minutes on three consecutive days to which the tubes had been exposed; and it is also true that mere microscopical examination does not suffice

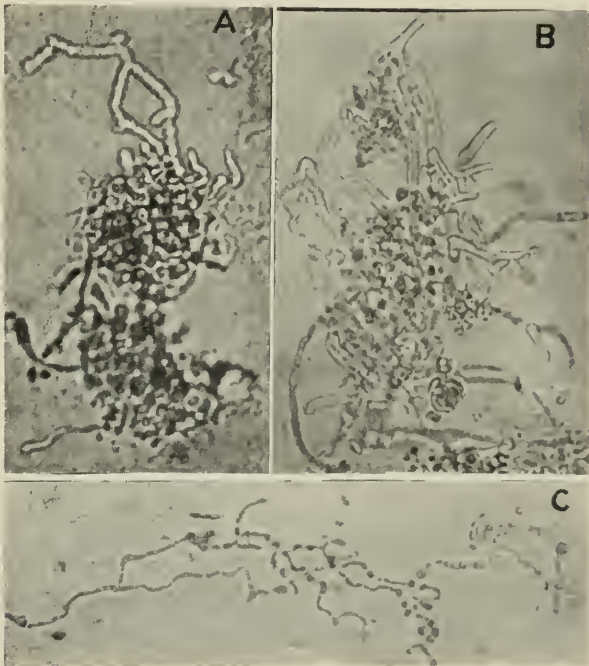


FIG. 3.

A, an incipient mould with very many germs ($\times 500$); B, a very delicate mould, with germs ($\times 500$); C, portion of another mould, lightly stained ($\times 375$).

for the discrimination of living from dead organisms. The organisms seen, or some of them, might be dead organisms.

It is here that tyrosine comes to our aid, since the use of a small quantity of a very dilute solution of this substance causes the living organisms that may be present to grow and multiply so freely as to dispose of objections (1) and (2) in the most unmistakable manner.

The organisms that are met with are produced very slowly and grow and multiply very slowly, so that three months is about the shortest time in which we may hope to find them within the previously sterilised

tubes, and in many cases it may be seven or eight months before they are large enough to be distinctly recognisable. Methods for cultivating the organisms have been tried and described elsewhere,¹ but they have not always been successful—often quite the reverse—so that I was most pleased to hear of the probable more certain influence of tyrosine.

Its use was suggested to me by A. H. Drew, whose researches, in conjunction with Dr. J. W. Cropper, into "Induced Cell Reproduction in *Amœbæ*" had convinced him of its great utility in this direction.² He suggested the use of a 0.05 per cent. solution to be added to the experimental solutions in the proportion of thirty drops to each fluid ounce—and this at two distinct periods in the course of my investigations—namely, in the *first* place to ripe tubes ready for examination; and *secondly*, by adding it from the first to the experimental solutions, in the hope that it might possibly suffice to much shorten the probation period needed before examination of the tubes.

Early last December I began to make such trials upon five series of tubes which were then fairly ripe for examination—that is, after intervals of seven to nine months from the dates of their sterilisation. The procedure adopted was of this nature: From each tube that was opened a sample of the deposit was taken with a well-sterilised pipette, and then with another pipette about twenty drops of the sterilised tyrosine solution were added.³ The tube was then securely closed, and again placed in the incubator or exposed to light.

After an interval of three or four weeks, for the most part, each tube was opened again, and another sample of its deposit was withdrawn with similar precautions. It was then, after mounting and staining with eosin or carbol-fuchsin, carefully examined so that what was found might be compared with the result of the examination of the earlier sample from the same tube.

The tubes that were treated in this way belonged to the five series whose constitution was recently described in a letter to the *British Medical Journal* (January 23, 1915, p. 183), and all of about forty of these tubes that have been examined have shown a very remarkable increase in the number, as well as in the size, of the organisms as a result of this addition of the tyrosine. The most fruitful solutions

¹ "The Origin of Life," 2nd edition, 1914, pp. 39-41, 79; and *Nature*, January 22, p. 581, and December 24, 1914, p. 466.

² See "The McFadden Researches" (Murray), vol. iv, April, 1914.

³ This solution requires to be guarded most carefully and re-boiled at intervals before use, as it is most prone to become contaminated with bacteria.

have proved to be Nos. 1, 4 and 5, though in Nos. 2 and 3 a smaller number and less varied organisms were to be found.¹

Figs. 4 to 8 show photomicrographs of some of the organisms found after the addition of tyrosine to the comparatively ripe tubes. Before

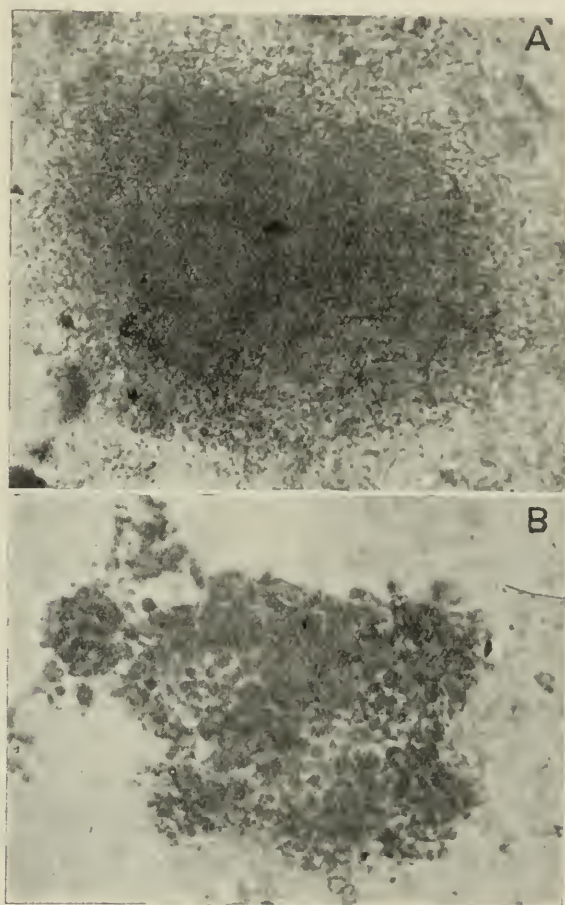


FIG. 4.

A, one of many dense masses of bacteria, stained ($\times 500$); B, a mass of multiplying germs, slightly stained ($\times 500$).

the tyrosine the organisms were mostly very small and not too numerous, having often to be very carefully searched out; while after the influence of the tyrosine there were no longer any difficulties of this kind, or

¹ The constitution of the three more successful fluids was as follows, the ingredients mentioned being those contained in each fluid ounce of distilled water. They were also added in the order mentioned. For No. 1, ammon. phos., 3 gr., dil. phosph. acid, 8 drops, and dil. sod. silicate, 1 drop. For No. 4, 1 drop of a 10 per cent. solution of iron sulphate and 2 drops of a 10 per cent. solution of potas. ferrocyanide. And for No. 5, the same two 10 per cent. solutions were used but in different quantities, thus—iron sulphate, 2 drops; potas. ferrocyanide, 3 drops, and sod. silicate, 5 drops. The dilute sodium silicate has been from a stock kept by Messrs. Allen and Hanbury, 6, Vere Street, London, W., from whom small quantities are still to be obtained.

indeed was there any room for still maintaining a sceptical attitude on the ground that what are to be found in my tubes are either dead organisms or mere simulacra: the unmistakeable growth and multiplication of the organisms effectually negative all such suppositions.

At the time that these trials were being instituted I prepared six and a half dozen new tubes charged with the same five kinds of experimental fluids, except that the tyrosine solution was added in the proportion of thirty drops to each fluid ounce. These sets of tubes were boiled for twenty minutes on three successive days, as the others had been, and were thereafter also placed in the incubator or exposed to light. The tubes containing No. 4 solution

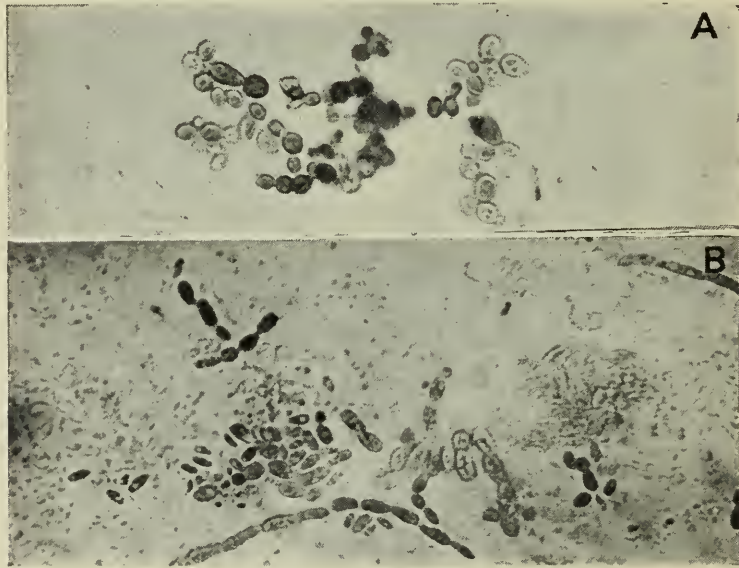


FIG. 5.

A, germs multiplying ($\times 500$); B, germs multiplying and developing ($\times 500$).
Both partly stained.

were all placed in the incubator, as I had previously found that they became partially decomposed when exposed to light.

After intervals of three to four months two or more representatives of each of the five series have been examined, and quite contrary to what had been hoped, every one examined was found to be barren. Not only were there no organisms detectable, the deposits also were found to be more scanty or distinctly different in character from what had been found in the respective tubes of earlier series in which there was no tyrosine. This latter fact seems to indicate some harmful influence of the tyrosine upon the solutions; and that it was not due to an alteration in the constitution of the tyrosine itself is shown by the

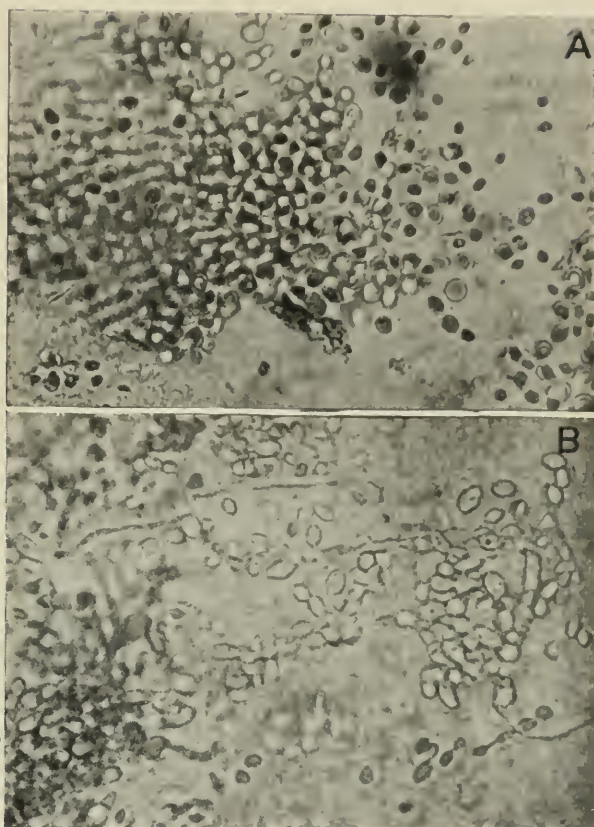


FIG. 6.

A, nucleated germs multiplying, partly stained ($\times 500$); B, torulae multiplying, unstained ($\times 500$).

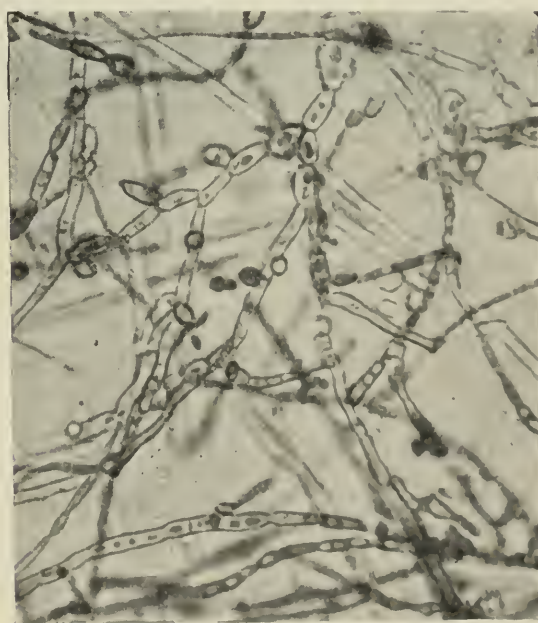


FIG. 7.

Portion of one of many moulds, showing hyphae and germs ($\times 500$).

fact that tyrosine thrice boiled for twenty minutes when added to ripe tubes has induced the same kind of multiplication and growth of organisms as when the tyrosine solution had only been boiled for a few minutes.

This unexpected sequence has, however, a very important bearing upon the general question, and tells strongly against the attempts to explain my results in either of the ways referred to under (3). The same materials were used as before in the solutions, and the deposits were examined in the same way, yet no organisms were seen or could be found, simply because of some altered constitution of the fluids produced by the addition from the first of a few drops of tyrosine.

The tyrosine, in fact, when added from the beginning has proved a potent agent for showing the groundlessness of the third category of objections; just as the effect of its subsequent addition to ripe tubes has shown in the most convincing manner that the other two suppositions are equally groundless as explanations of my results. Mere simulacra or dead organisms may be not altogether absent; but with the help of the tyrosine it has been positively shown that the tubes contain crowds of living organisms of different kinds.

It is much to be desired that some skilled bacteriologists and fungologists would now take up this inquiry, and cultivate the organisms found by the most approved methods, so as to let us know to what extent they are new and to what extent they correspond with forms already well known. Many of them will probably belong to this latter category. Some of the moulds, for instance, that I have found seem to be of *Streptothrix*, and others of *Penicillium* type; while George Masee has pronounced others to be of *Oöspora* and *Cladosporium* type.¹

This very fact is felt by some to be a strong reason for still retaining a lingering suspicion that there must have been some flaw in the experiments. Such objections may, however, it seems to me, be fairly met by recalling what is known concerning crystallisable, as opposed to living matter. We know that the former may show itself in one or

¹ The latter is a remarkable mould recently found in tubes, only just over three months old, of the No. 4 series. I have seven of these tubes, and when I opened one of them on March 29 I found several specimens of such a mould as I had never previously seen. Two days after I opened another, and found the same kind of mould. On April 5 Sir William Ramsay called to see me, and as he had previously expressed some scepticism about my results, I opened the third of these tubes in his presence, telling him that I felt sure we should find in it the same kind of mould that had been taken from the other two tubes. And this prediction was verified. Yet these tubes had been prepared from the same 10 per cent. solutions that had been used a few days previously for the other No. 4 series to which tyrosine had been added from the first, and which had been found to be barren.

other of some 230 different forms, some of which are rare and others very common. Supposing a chemist after much labour has succeeded in producing synthetically in his laboratory some very important new product, would he, or would his colleagues, doubt the reality of what he had achieved simply because it was found that his new product had assumed the form of one of the commonest and most familiar of the crystalline types?

If we contrast the comparative simplicity of crystallisable matter with the myriad modifications in molecular constitution possible in living matter, we may find some possible explanation of the almost



FIG. 8.

One of many more developed moulds, stained, found three months after the addition of tyrosine ($\times 300$).

endless varieties of bacteria and of simple moulds. And if, as our experiments would seem to indicate (in view of the uniformity of natural phenomena), living matter has ever been coming into being in suitable media since its first appearance on the surface of our Earth, why may they not also have been appearing in multitudinous forms belonging to one or other of these common types, which, as we know, are now to be met all over the surface of the Earth? If this has been the case, then the appearance of such common forms within our experimental tubes would be much less difficult to understand. An extension of the common point of view would, of course, be needed. We should have to

believe that *the ubiquity of these common forms is due to continued new origins, as well as to their dissemination by reproductive processes.*

A related almost insuperable difficulty in the minds of others arises simply from the existence of a long-cherished preconception. They find it impossible to believe that such organisms as bacteria and simple moulds can be products of so-called "spontaneous generation." This difficulty was expressed very strongly by Huxley in 1870, and is undoubtedly still felt by many. But after all, what does the objection come to? Preconceptions are cherished and set against the potencies of Nature. That is all. They say it is not conceivable that such organisms could appear as products of *de novo* births. By which they can only mean that they are quite unable to explain it. But multitudes of facts have to be admitted which are similarly or much more inexplicable. If it were not well known and established that a simple cell, a fertilised human ovum, may be able to develop into a man or a woman with a mixture of physical and mental attributes common to its ancestors, would not that be far more inconceivable? Yet no well-informed person doubts that this marvel, utterly inexplicable in all its details (that is inconceivable), is ever being repeated.

Let us come back for a moment from the region of preconceptions and face other facts. The sceptics now know that such and such organisms are to be found within my experimental tubes, although some months previously these had been heated to 100° C. for twenty minutes on three successive days. If they still doubt that such organisms can arise as products of so many new births, the only position tenable by them, from the point of view of science, is to contest the fact that such organisms would be killed at the initiation of the experiments by the sterilising processes. This, however, they do not attempt to do. They know quite well, in fact, that *the majority of bacteria, that torulæ, and moulds, and their spores generally, would all have been killed by the very first of these three boilings.*

After some of my specimens of Oöspora had been sent to Kew for identification, the Assistant Director, writing on January 15, 1913, said: "As far as we can ascertain, no fungus spores have ever been known to survive heating to the temperature and under the conditions indicated in your letter." Subsequently a mass of this mould about $\frac{1}{2}$ in. in diameter was seen growing within one of my unopened tubes.¹ While, in reference to the specimens of Cladosporium recently sent to him, George Masee, *facile princeps* of fungologists, wrote: "I am very pleased

¹ See *Nature*, January 22, 1914, p. 581.

to have seen the specimens, and as they have appeared after the drastic treatment of the media you describe, it compels us to pay attention to the subject and to look upon the origin of life from a new standpoint."

Such knowledge as we have of all the marvels following upon the fertilisation of human ova is unfortunately denied us in regard to new births of living matter. It is futile to say, as so many have said, that such new births are contrary to the experience of all mankind, seeing that, from their very nature, they must in their incipient stages always remain altogether outside the pale of human experience. As I have elsewhere lately said,¹ "Archebiosis is a process that always takes place beyond our ken, seeing that it must begin with mere molecular collocations, gradually going on to the formation of particles of an ultra-microscopic order. And the only explanation that seems possible of the growth of such particles into organisms like those found in the tubes, as well as of the appearance of the heterogenetic products that we have seen proceeding from zooglœal segments, is to fall back upon an explanation which is generally admitted to account for all the known forms of crystalline matter. *Molecular constitution combined with the influence of the environment* is what we have to appeal to there; and as Herbert Spencer over and over again insisted, the forms and structures of organisms, under the influence of what he termed 'organic polarity,' must be dependent upon like causes. In accounting for the lower forms of living matter, therefore, we may suppose, as he says,² that their 'organic molecules of each kind, no matter how complex, have a form of equilibrium in which, when they aggregate, their complex forces are balanced.' "

The foregoing quotation is from a communication to *Nature* entitled "The Production at Will of either Fungus-germs, Flagellate Monads, or Amœbæ from the Ultimate Segments of Small Masses of Zooglœa." It deals with instances of heterogenesis of great importance, which, while showing the community of origin of members of both the Animal and the Vegetal Kingdoms from aggregates of Bacteria, affords also what appears to be a striking exemplification of the view above indicated that in reference to the forms and natures of lower organisms "molecular constitution combined with the influence of the environment" are the essential and dominating factors.

In a presidential address to the Quekett Microscopical Club last year

¹ *Nature*, December 24, 1914, p. 467.

² "Principles of Biology," revised edition, 1898, vol. i, Appendix D, p. 704.

Professor Dendy, after referring to some of my results with the tube experiments and the apparent *de novo* origin of moulds, says: "I must confess that I myself find it impossible to believe, without much stronger evidence, that such comparatively highly organised beings can have been evolved so rapidly from ultra-microscopic germs." Further on he also says: "Dr. Bastian himself, it should be observed, is a convinced upholder of the doctrine of heterogenesis, or the sudden appearance of one kind of organism as the offspring of another, but it may be doubted whether any other living biologist holds similar views."

I trust that Professor Dendy and other doubters may find the additional evidence required in proof of Archebiosis in the present communication, and similar easily verifiable proof of the reality of Heterogenesis in that above referred to which was published in *Nature* near the end of last year.

Demonstration of Methods for the Isolation and Identification of *Bacillus typhosus* and Allied Organisms.

By GEORGES DREYER, M.D., E. W. AINLEY WALKER, M.D.,
and A. G. GIBSON, M.D.

THE light method of isolating particular micro-organisms devised about ten years ago by Dreyer, and the technique for quantitative agglutination tests with standardised killed cultures introduced by him about the same time, were described.

They have been applied by the authors in the investigation of cases of typhoid and paratyphoid infection (including carriers) at the Third Southern General Hospital at Oxford, with results which have already been published in the *Lancet*, February 13 and March 27, 1915. Agglutination tests carried out in the manner described afford the most delicate and conclusive method of bacteriological diagnosis in suspected cases of active typhoid or paratyphoid infection. And so far as evidence at present goes, it seems probable that the light method will prove a valuable addition to our methods of isolation, particularly in the detection of typhoid and paratyphoid carriers.

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(2) *Agglutination Method:*

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Section of Psychiatry.

January 26, 1915.

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

The Application of Physiology and Pathology to the Study of the Mind in Health and Disease.

IN his illuminating work on Mental Physiology Dr. Maudsley thus comments on the subject which I have ventured to take for my address to you this evening, in the chapter, "The Method of the Study of Mind," p. 48:—

"The past history of Psychology—its instinctive progress, so to speak—no less than the consideration of its present state, proves the necessity of admitting the objective method of the study of mind. That which a just reflection teaches incontestably, the present state of physiology illustrates practically. Though very imperfect as a science, physiology has made sufficient progress to prove that no psychology can endure except it be based upon its investigations. Let it not, moreover, be forgotten (as it is so apt to be) that there is continuity throughout Nature, and the divisions in our knowledge are artificial; that they should be accepted and used rather, as Bacon says, for lines to mark and distinguish than sections to divide and separate; in order that solution of continuity in sciences may always be avoided."

This was written nearly fifty years ago, and physiology has made notable advances, yet one must still agree with the further statement of Maudsley that no one pretends that physiology can for many years to come furnish the complete data of a positive mental science. We can, however, realise that physiological science has made great advances in its application to the study of mind, and in no direction more important than in the bio-chemistry of the ductless glands, and the influence of their internal secretions (hormones) on the bodily and mental functions in health and disease, an addition to knowledge which

we owe in greatest measure to English physiologists. It will be my endeavour this evening especially to direct your attention to the desirability of the application of this branch of physiology to psychiatry. This influence of internal secretions on the mind was alluded to by Maudsley in his "Physiology of the Mind," and it is interesting to see how this modern development of the hormone theory is anticipated by him in the following lines:—

"It is probable that many vague feelings or indefinite emotional states to which we have no adequate or corresponding ideas are produced by the operations of the internal organs; they are of a very vague character, and cannot be expressed in definite objective signs, wherefore they cannot become knowledge. We have the best instances of what I mean in the vague, overmastering feelings of pregnancy, and those to which the development of puberty gives rise; and it is certain that we must go deeper than self-conscious analysis will ever get, to arrive at their true nature and causation. Even the passion of love itself has its source in the unconscious life and can no more be explained in consciousness than the feelings of hunger and thirst; it marks an elective affinity in the organism which oftentimes enslaves consciousness and overpowers volition."

There is reason to believe that each internal organ of the body has its representative centre in the supreme cerebral centre, through which it takes its essential part in the constitution and function of the mind. Maudsley further says:—

"Now the brain is the leading member of this physiological union, the centre in which the different organic functions meet and are co-ordinated; wherefore the supposition that it carries on this important function of organic life quite apart from and independently of its function as the organ of mind, would be most improbable, even if observation did not contradict it. As a matter of fact, observation does contradict it positively. We have the plainest instance of this in the case of the reproductive organs, the functional development of which, taking place somewhat abruptly at puberty, works a complete revolution in the mental character. The individual is transformed, his entire sentiency is changed, and he becomes susceptible to impressions which before were completely indifferent to him: a look, a tone, an odour, a touch arouses an emotion which is quite new to him, and sympathetic ideas come he knows not whence or how. Strange and vague feelings, aimless longings, obscure impulses and novel ideas witness to the commotion which the newly developed function is making by its eruption into the mental life; there is an awakening of sensual impulses which clothe themselves in mental forms, of mental necessities which clothe themselves in sensual images. It is now, too, that altruistic feeling begins to germinate in the mind; before puberty a boy is the most complete egoist, taking as a matter of course all the affection and care

which are lavished upon him; but after puberty he begins for the first time to have some sense of what others do for him, and to display some feeling of his obligation to them. These evolutionary effects of the functional development of the reproductive organs do not take place when such development is prevented by their removal before puberty."

What a splendid analysis Maudsley has thus given of the complete revolution of the mind at puberty, and how well he has indicated the importance of a full investigation of the histology of the sexual glands in health and disease in relation to the study of insanity.

Nature is unmindful of the individual, mindful only of the species, and the sexual impulse is the strongest of all physiological impulses. It is true that at the dawn of the passion "love is blind." It is blind because the sexual impulse is excited by a bio-chemical stimulus derived from the internal secretion of the sexual glands, arousing vague instinctive desires which, as Maudsley says, have their source in the unconscious life, and which work a complete mental revolution. But if love is blind, it is also true that "love adds a precious seeing power to the eye." This is apparently paradoxical, but the latter is not a biological antithesis to the former, for the latter implies a perceptual instinct of sexual attraction necessary to materialise the bio-chemical stimulus and effect the preservation of the species. That this perceptual attraction is instinctive is shown by the fact that the language of love is universal; it cannot be concealed where it exists; it cannot be feigned where it does not exist; it appeals by mute eloquence in expression, attitude, and eye in a manner more enticing and forcible than any spoken language. Yet the sexual impulse is the one that is subjected to the greatest amount of repression on account of the mental attitude of society, and for that reason it has always been one of the weakest points in our cultural development. Freud and his school assert that feelings of a sexual nature appear before puberty, and the newborn child brings with it the germs of sexual feelings, which continue to develop for some time, and then succumb to a progressive suppression, which is in turn broken through by the proper advance of sexual development, and which can be checked by individual idiosyncrasies. I do not propose to discuss the Freudian doctrines here, but later I shall bring forward some evidence of a histological nature in support of the above statement.

It must have struck everybody that has had to do with insanity that there are two periods of life when its onset specially occurs—namely, early adolescence and the involutinal period. In the study of the

relation of heredity to insanity I was particularly struck by this fact. Now are we to assume that the mental disorder of the true insanities or psychoses, even those which terminate in dementia, as dementia præcox, can be explained by structural changes of the brain, macroscopic or microscopic? Or should we not rather believe that they are either the result of microbial toxic conditions of the blood, or, where this cause is improbable, due to disturbance of the physiological equilibrium of the internal secretions of the ductless glands, which experiments on animals and clinico-anatomical investigations in man have shown play an all-important part in the healthy functioning of body and mind? Moreover, we have in recent years learnt that the interstitial gland structure of the sexual organ can function independently of the genetic gland structure; and there is abundant proof to show that functionally correlated with the sexual glands are the thyroid, the parathyroid, the hypophysis, the pineal gland, the cortical portion of the suprarenal gland, the thymus, and probably also the islands of Langerhans of the pancreas. Therefore, to study the relation of a disorder of the internal secretions to various forms of insanity it is necessary to make a systematic examination of all these glands in health and disease at various age-periods from birth onwards; for this purpose it was necessary for comparison to collect material and obtain the various organs from hospital and asylum cases, dead from injury, and acute and chronic bodily disease accompanied and unaccompanied by mental disorder. An investigation of this material in a systematic manner must be made before any just conclusion can be arrived at in respect to the correlation of a disturbance of equilibrium of the internal secretions of the reproductive gland system and insanity. Already I have observed a remarkable variation in the weight of the thyroid gland, especially in women, 4.1 grm. the lowest and 51 grm. the highest.

HYPOTHYROIDISM IN RELATION TO THE SEXUAL GLANDS AND HORMONE EQUILIBRIUM, AS A CAUSE OF BODILY AND MENTAL DISEASE.

I have already obtained many objective indications pointing to close association of mental disease with disease of the ductless glands; thus I brought before this Section and published, in conjunction with Dr. Brun, an account of "A Microscopical Investigation of the Nervous System in Three Cases of Spontaneous Myxœdema."¹ One of these was a case dying in Claybury Asylum, of special interest because it

¹ *Proc. Roy. Soc. Med.*, 1913, vi (Sect. of Psych.), pp. 75-100.

showed a marked increase of lymphoid and fibrous tissue with nearly complete absence of colloid in the thyroid gland. There was a substitutive increase of colloid in the parathyroid, and especially an increase of colloid of the *pars intermedia* of the pituitary. The objective signs of myxœdema were slight in this case when the marked destructive change of the thyroid glandular substance is considered, but the mental disorder was pronounced. She died of heart failure, with a pulse-rate of only 40. Examination of the central nervous system showed marked chromolytic changes of the nerve cells, affecting in a particularly grave manner the autonomic bulbar system—viz., the nuclei of the ninth and tenth nerves, and in a less degree the cerebrospinal motor neurones, and the sympathetic system. Dr. Brun and I called attention to the comparatively frequent coincidence of the myxœdematous syndrome with an acute psychosis, starting suddenly (as in two of the cases we reported), and having for the most part the characters of a melancholic or a manic-depressive insanity.

THE BIO-CHEMICAL INTER-RELATION OF THE SEXUAL GLANDS AND THE DUCTLESS GLANDS.

There are many facts pointing to the bio-chemical inter-relation of the sexual glands and the ductless glands; thus it is known that the thyroid glands increase in size during pregnancy; there is, indeed, a distinct hypertrophy occasioned by a marked increase of colloid in the follicles. The development of the corpus luteum and the passage of its hormone into the blood is probably the cause of the hypertrophy. An increase of the thyroid occurs at puberty, during menstruation and at the climacterium. Changes also occur in the parathyroids during pregnancy. In the anterior glandular portion of the hypophysis, certain cells which normally occur in the gland are greatly increased and constitute the so-called pregnancy cells, which structural increase is partly the cause of the increase of weight of the gland in pregnancy. A large increase of the lipid cholesterol ester content of the cortical cells of the adrenal gland occurs, and it is of interest to note that these cells structurally and chemically closely resemble the lutein cells of the Graafian follicle. Indeed, it has been suggested, and there is considerable evidence in support thereof, that we must not only look to a toxæmia caused by derangement of the function of the liver and kidneys to explain the occurrence of vomiting, eclampsia and mania in pregnancy, but also to the probability that these pathological conditions may result

from a disturbance of the physiological equilibrium of the internal secretions. Again, this disturbance of the balance of the internal secretions which is liable to occur at the menopause may be the cause of the frequent occurrence of psychoses and neuroses of various kinds so frequently met with in women for the first time. I shall later, when referring to the development of the sexual glands, point out other facts in support of the bio-chemical functional inter-relation of the sexual and ductless glands.

OBSERVATIONS MADE UPON THE SEXUAL GLANDS IN RELATION
TO INSANITY.

Since the war began I have made all the post-mortem examinations myself instead of having them done by my assistant, and I have been in the habit of examining all the ductless glands. In conjunction with Staff-Surgeon Kojima, who has carefully weighed and preserved the glands, we have obtained some valuable material for the pursuit of this investigation of the relation of the sexual and ductless glands to insanity. I have sometimes found, after removing the brain, that the pituitary gland is enlarged, and then I have found that the thyroid shows either fibrotic atrophy or the gland substance has been replaced by fibroid and lymphoid tissue; and I may here remark that the thyroid may be of normal size, and yet contain no glandular substance; so that microscopic examination is necessary to reveal the extent of the disease. When I speak of enlargement of the pituitary I do not mean a tumour. I have frequently observed in cases of hypothyroidism that the ovaries have appeared fibrotic and atrophied, or have undergone cystic degeneration, as happened in the following case:—

A married woman, aged 57 on admission, who was said to be a dancer. Her speech was said to be slow and indistinct; she had tremors of face, hands and tongue. The memory was impaired and she had no idea of time or space. She was very depressed and attempted suicide before admission. She remained emotional and depressed and became demented, so that before death, nine years later, she was regarded as suffering from general paralysis. At the post-mortem examination, which I made a few days ago, I found no signs of brain disease apparent to the naked eye; it was certainly not a case of general paralysis. I observed that the *sella turcica* was very large, and the posterior clinoid processes were absorbed; the gland weighed 1.2 grm., which is more than twice the normal weight, and it was

especially the posterior lobe and *pars intermedia* that was increased so much in size. As I expected, I found the thyroid very atrophied and fibrotic, weighing only 12·6 gm. The interesting feature of the case was the finding of an infantile uterus; the left ovary completely destroyed by an old salpingitis, and the right one atrophied and damaged to such a degree that it weighed only 0·4 gm. I consider it probable that this patient early in life had suffered from gonorrhœa, which had caused ovarian destruction, atrophy of the uterus, and at the climacterium hypothyroidism, with substitutive increase of the colloidal *pars intermedia* of the pituitary. Microscopic examination of the thyroid gland showed a marked fibrosis and atrophy of the glandular substance; there was still some colloid present. In one of the cases of myxœdema which presented during life very pronounced objective and subjective signs and symptoms, I found a somewhat similar condition of the gland. In another case—that of a patient admitted to Claybury—who on admission showed signs of myxœdema, but died eight years later without the objective signs and symptoms being pronounced, but from the first had very marked mental symptoms, I found at the autopsy, made a few weeks ago, a large thyroid. This on microscopic examination exhibited only fibrous and lymphoid tissue, the gland was almost completely destroyed, and the pituitary gland was nearly twice the normal weight.

Now it is possible that the increase in size of the pituitary gland may have an important bearing in the production of the mental symptoms, for it is known that the colloidal substance secreted by the *pars intermedia* escapes through the infundibulum and the neurohypophysis into the cerebrospinal fluid. What part this internal secretion precisely plays we do not know; but if the cerebrospinal fluid serves as the lymph of the brain, as I believe it does, then the excess of this colloid pouring into the fluid which irrigates the perineuronal spaces may have an important influence upon the functions of the organ of mind. However, a great deal of work will have to be done before this can be more than suggested as a possible working hypothesis.

SYSTEMATIC EXAMINATION (BY DR. LAURA FORSTER) OF A LARGE NUMBER OF OVARIES IN DIFFERENT FORMS OF MENTAL DISEASE.

For years I had been struck with the fact that the ovaries of women suffering from insanity at an early age, and dying in early or late adolescence, have, at the autopsy, been found to be fibrotic. Knowing as we do that amenorrhœa is frequently met with in such young

women, I thought it would be desirable to have a systematic investigation made of the ovaries, and Dr. Laura Forster undertook and completed an investigation of the ovaries by serial sections in a hundred cases, for the most part obtained from persons who had died in the asylums, but also some at varying ages who had died in hospitals. As regards dementia præcox, she found that the ovaries of all those who had reached the age of 30 showed signs of early involution, by an increase of interstitial connective tissue, and there was a great scarcity of Graafian follicles. Even in those much below this age there was a distinct diminution of the follicles as compared with a normal woman of the same age. In imbecility with or without epilepsy the ovaries did not present a uniform type. Some quite young persons showed early involution, while in others there were a proportionately large number of follicles. In mania depressiva she found a diminution in the number of follicles. Thus in the ovary of a woman, aged 23, there were signs of the ovary undergoing cystic degeneration, and a marked increase of fibrous tissue. In general paralysis, women are not virgins, because, except in the case of the juvenile form, they are the subjects of acquired syphilis, and the mental disease is the result of infection of the brain. The ovaries of young women dying of this disease do not show early involution as a rule.

The results of this careful and laborious investigation of the ovaries in relation to mental disease are in a measure nullified for the following reasons: Dr. Forster herself showed that a young woman dying of chronic heart disease in a hospital, presumably without any mental affection, exhibited fibrotic changes in the ovaries similar to those observed in cases of primary dementia of adolescence. Dr. Forster has not separated her asylum cases dying from some acute bodily disease from those who have died of tuberculosis, or some other chronic disease usually met with as a cause of death in the subjects of dementia præcox. Consequently we do not know whether the changes described are due to the chronic bodily disease, or are to be associated with the mental defect. In her paper, which I hope to publish later, she has laid considerable stress upon the experiments of Ceni upon birds, in support of the correlation of dementia præcox with changes found in the ovaries. He removed one hemisphere, and the birds surviving the traumatic shock were killed after varying periods of a few months to three years, and their ovaries were subsequently examined histologically. The primary shock had the effect of causing them to cease laying eggs for some months. In the following year they began to lay again, but

in the second year fewer eggs were laid, or the birds ceased altogether from laying. The birds were otherwise in a healthy condition. The examination of the ovaries showed a premature progressive involution. Ceni concludes that there are intimate relations between the brain and the ovary. It would have been interesting if Ceni had conducted similar experiments on male birds, and proved that lesions of the brain can produce a premature dynamic exhaustion of the testis. And that leads one to say that Dr. Kojima and I are systematically investigating the male reproductive system and ductless glands. For if an abnormal condition of the sexual glands and of the internal secretions of the ductless glands, causing an hormone failure or disorder of the hormone equilibrium, has a causative relation to dementia præcox, it should be possible to establish a structural defect or change in the corresponding reproductive organs and ductless glands of the male. The work of Miss Forster at present, therefore, may be regarded as highly suggestive, but not affording convincing proof. The work she has begun, however, will now be extended in a systematic manner by Dr. Kojima.

I was fortunate enough recently to obtain a case of dementia præcox who died of pneumonia after a few days' illness, and whose bodily condition prior to this, judged by the appearance of the organs, must have been sound. The ovaries in this case were fibrotic and showed very few follicles or corpora. So that it seems probable that there may be some correlation, if not causative relation, between the hypo-function of the ovaries and an insanity of adolescence terminating in dementia. Seeing that the sexual glands are fundamentally the *fons et origo* of the passions, a hypo-function of them might, by the absence of the hormones, account for the *emotional* indifference so characteristic of this form of mental disease.

Again, my investigations of insane parents with insane offspring show that the children of insane parents when they become insane do so in a large proportion of cases in adolescence. Anticipation thus tends either to elimination of the unsound members by bringing it on at an earlier age or by a diminished durability of the germinal cells.

MICROSCOPICAL EXAMINATION OF THE SEXUAL GLANDS FROM BIRTH ONWARDS.

An examination of the ovaries and testes at different ages from birth onwards has proved to be very interesting from several points of view. In that admirable text-book, the "Principles of Human Physiology," of Professor Starling, ovulation is thus described (p. 1366):—

“At birth the ovary consists of a stroma of spindle-shaped cells, and is covered by a layer of cubical epithelium (the germ epithelium) continuous with the epithelium lining the general peritoneal cavity. Embedded in the stroma, but especially numerous just underneath the epithelium, are a vast number of ‘primordial follicles.’ These are formed during the foetal life by down-growths of the germinal epithelium. Of the cells prolonged in this way from the germinal epithelium, some undergo enlargement to form the primordial ova, while the others are arranged as a single layer of flattened nucleated cells, the ‘follicular epithelium,’ as a sort of capsule to the ovum. Of the primordial follicles, about 70,000 are to be found in the ovary of the newborn child. During the first twelve to fourteen years of life they remain in a quiescent condition. With the onset of puberty one or more of the follicles begin to develop. Indeed, this development may be regarded as the causative factor in the various phenomena which are characteristic of puberty in the female (*vide* p. 1361). The first stage in the growth of the follicle is a proliferation of the follicular epithelium, the cells of which become cubical and are arranged in several layers around the ovum. At one point in the mass of cells surrounding the ovum a cavity appears filled with fluid—the *liquor folliculi*. The epithelium thus becomes separated into two parts—i.e., the *membrana granulosa*, several layers thick, lining the whole follicle, and the *discus proligerus*, a mass of cells attached to one side of the follicle, in which is embedded the ovum. Round the growing follicle the stroma assumes a concentric arrangement and forms a capsule, of which the internal layer consists chiefly of spindle-shaped cells richly supplied with blood-vessels, while the outer layers, the *theca externa*, is made up of tough fibrous tissue. With the growth of the follicle the ovum also becomes larger and surrounds itself with a distinct membrane, the *zona pellucida*.”

It will be observed that Professor Starling considers that the formative growth of a follicle does not occur until puberty. But I have a microscopic drawing of a section of the ovary of a child aged 8 days, and one of a child aged 1 year 3 months, which shows that even a few days after birth the primordial follicles are commencing to undergo development into Graafian follicles and the ovum is being surrounded by actively developing epithelial cells which will later on form the *membrana granulosa*; outside of this are seen large connective tissue cells which will form the *theca interna*, constituting the interstitial gland of Limon. The section of the ovary of the child aged 15 months shows a Graafian follicle quite as well developed in every respect as that described and figured by Professor Starling. Seeing that there are 70,000 ova to be found in the ovary of the newborn child, and during the period of life when pregnancy can occur (12 to 48) not more than 400 follicles can rupture at the menstrual periods, of which there is a possibility of relatively few being fertilised, there is then an extra-

ordinary prodigality of primordial follicles. We cannot, however, believe that the primordial follicles ripen and form Graafian follicles in early life without effecting some useful purpose. Should we regard the epithelial cells of the *membrana granulosa* or the proliferated interstitial cells of the *theca interna* as the source of the hormone that fixes and maintains the secondary bodily and mental sexual characters? The balance of evidence is in favour of the thecal cells secreting the hormone that fixes the secondary sexual characters, and of the view that the development of the Graafian follicles leads to a formative proliferation of the thecal cells in the stroma of the gland. The ovum does not escape from these follicles developed in early life, but degenerates. Subsequently an invasion of the fibrous stroma occurs to form the atretic follicle. I have a photograph of an atretic follicle from the same section of the ovary of a child aged 18 months, also from the ovary of a congenital imbecile and a case of melancholia.

Sections of the testis at various ages prior to puberty show the seminiferous tubules surrounded by an interstitial tissue. The tubules are lined by cells which are called the syncytium of Sertoli, and within this layer of cells are the generative cells, the spermatogonia from which the spermatozoa are developed. There are many reasons for asserting that the internal secretion which fixes and determines the secondary mental and bodily male sexual characters is derived from the interstitial cells (the cells of Leydig as they are called), and which are homologous with the cells of the *theca interna* of the ovary. Among the reasons which may be mentioned are the following facts: (1) Cryptorchids possess the secondary sexual characters, although there is default of development of the generative structure of the gland. (2) Destruction of the generative structure of the ovary and testis by X-rays can be brought about without destruction of the interstitial gland structure; nevertheless, sexual desire and the secondary sexual characters persist.

If we examine the ovary when the genetic function has commenced, a bio-chemical change may be noticed. In the ovary around the follicles that have ruptured are seen the characteristic lutein cells, which in unstained sections appear as pale yellow cells, due to a lipochrome substance similar in composition to the yolk of an egg. If these sections are stained with Scharlach or Sudan III they stain deep red owing to their being full of granules of a fatty cholesterol ester lipid. I have some coloured drawings which illustrate this appearance. I am not certain whether these lutein cells arise mainly from the interstitial cells or from the epithelial cells of the *membrana granulosa*. They no doubt play an

important part in the development of the ovum and in the formation of the internal secretion of the corpora lutea. Sections of the ovary prior to puberty show little evidence of lipoid-containing cells; neither do sections of the testis. But, after puberty, not only are the interstitial cells of Leydig loaded with this lipoid, but the lipoid can be seen within the cells constituting the generative structure of the gland. I have a drawing of a section of a seminiferous tubule of a soldier, aged 20, who died from the effects of a bullet wound of the brain in Charing Cross Hospital. It is remarkable that this gland shows little or no evidence of active spermatogenesis. Has the injury to the brain arrested the generative process? All the tubules show an abundant accumulation of lipoid substance, and it is permissible to suppose that this is the raw material out of which the spermatogonia manufacture the nucleinic acid which forms the main constituent of the head of the spermatozoon.

In Carpenter's "Mental Physiology" there is a remarkably interesting chapter on Habit, which is extensively quoted by William James in his "Principles of Psychology." This chapter commences with the following statement:—

"There is no part of man's composite nature in which an intimate relation between Mind and Body is more obvious than it is in the formation of habitual modes of activity, whether psychical or corporeal; the former, like the latter, being entirely conformable to the laws which express the ordinary course of the instinctive operations. In the first place it is characteristic of every living organism to build itself up according to a certain inherited type or pattern, so that we must attribute to its germ a formative capacity in virtue of which it turns to account both the food and the force which it derives from without."

When we reflect that 27,000,000 spermatozoa may be discharged at a single coitus, each one carrying the characters of species, race, and the individual characters of ancestors, we may feel some hesitation in accepting the chromosome or particulate theory of inheritance, and seek for some bio-chemical process by which the male germ cells possess this unlimited power of building up specific living matter.

Ostwald has shown that the heads of the spermatozoa contain a nucleinic acid which is common to all animals, but that each species has a specific protamin ferment, and it may be that this specific protamin is capable of "this formative capacity in virtue of which it turns to account both the food and the force which it derives from without."

PRECOCIOUS PUBERTY.

It is well known that normal individuals, male and female, may show sexual impulses in early life, and, years before the normal time for puberty, develop the secondary sexual characters. And particularly does this early onset of the reproductive function happen to female natives of hot climates. Apart from this physiological condition there have been recorded a number of remarkably interesting pathological cases of *pubertas præcox*, occurring in both sexes as a result of tumours of the testis, ovary, and pineal and suprarenal glands, which are of great interest in respect to the influence of the internal secretions upon the secondary sexual characters.

Biedl, in his great work on the "Internal Secretions," cites some interesting cases. One was that of a boy reported by Sacchi, who was completely developed as regards the bodily and mental sexual characters at the age of $9\frac{1}{2}$. He was 143 cm. in height, weighed 44 kilograms: he had a long black beard and pubic hair well developed. The left testis was greatly enlarged by a tumour which was, on removal, found to be an alveolar sarcoma; following the operation there was a regression of the secondary sexual characters. He also quotes an analogous case in a girl aged 6, where a sarcoma of the ovary had induced a precocious physical and mental development and a menstrual flow. After removal the menses ceased, the hair on the pubes and in the axilla fell out, the mammary glands regressed, and the child-like characters returned in every respect except that of the voice.

Now the pineal body is a glandular structure which in adolescence undergoes regressive changes. We do not know whether these tumours in which precocious puberty occurred were due to hypertrophy of the glandular tissue or were due to a growth which caused its destruction. But Biedl states that basal tumours of the pineal gland of the nature of *teratomata* have been reported as occurring in individuals under the age of 7, who, besides being affected by symptoms of pressure upon the *corpora quadrigemina*, have presented symptoms of precocious mental development. The secondary sexual characters were developed at an early age. In the Department of Neurology at the Cornell University "The Functions of the Pineal Gland" have been recently studied by Dana, Berkeley, Goddard, and Cornell, and in the report which they publish they summarise in the form of a table the pathological and physiological effects attributed to the activities or disorders of the pituitary body and the pineal body. It seems that whereas tumours

of the pituitary are associated with adiposity, sexual changes, genital atrophy, and infantilism, tumours of the pineal gland are associated with early development of the sexual organs and bodily functions, and early bodily and mental maturity.

The authors also report the feeding of animals and mentally defective children at Vineland. In respect to the latter investigation, they claim only a presentiment of a direct influence of the extract towards mental improvement, and conclude that "all such experiments are subject to so many disturbing factors that it seems necessary, in view of the slight gain recorded, that the experiment should be continued for a longer time.

THE ADRENAL GLANDS.

One of the most important researches on the internal secretions of the ductless glands, of the many which we owe to the English physiologists, relates to the functions of the suprarenal glands. It has been known since the time of Addison that this gland is essential to life, and I need not tell you what is well known regarding the function of this gland in virtue of the adrenalin which it secretes, except in so far as it has been shown by Cannon and Elliott that the emotion of fear through the splanchnic nerve liberates adrenalin in excess into the blood. The whole story of the adrenal glands has recently been so admirably told in the Sydney Ringer Lecture, that I should recommend all who are interested in the functions of the ductless glands to read it. Still, in support of my thesis of the application of physiology to the study of mental processes, I cannot refrain from quoting one or two important generalisations from that lecture¹:—

"Morphology, therefore, tells the same tale as physiological analysis. The adrenalin cells and the sympathetic nerves belong to a common system, whose first duty is that of sustaining the activities of the circulatory muscles. As the animal develops its muscular efficiency, learns a hundred new functions, and with a constant body temperature becomes independent of its environment, the sympathetic system becomes more and more complex and is split up into manifold possibilities of delicate adjustment. All these are but refinements of means for the one great end—to enable the animal to move more swiftly, to catch its prey and to fight. Fighting power rises with rise of the blood-pressure, reserves of sugar to feed the muscles are hurried up from the liver on the call of the circulating adrenalin, the daily routine of digestion is checked by intestinal inhibition, and the various segments of the bowel are cut off from one another by the closure of the sphincters. Cannon has reiterated this

¹ *Brit. Med. Journ.*, 1914, i, p. 1394.

view and, further, shows very neatly how the clotting of the blood by adrenalin which has been observed by myself and others is a further elaboration to check leakage in any chance wound of the body during action.

“By that curious antithesis of the emotions upon which Charles Darwin laid stress, the machinery employed to prepare for fight may, with the cowardice of civilization, be set as powerfully in motion only to express fright. And in this the adrenalin is equally, or perhaps even more, exhausted.”

Our knowledge of the function of the cortex of the gland is still obscure. I have observed that in the systematic examination of the ductless glands the cortex has varied greatly in amount in different cases; and microscopic examination has shown, after staining with Scharlach, that the amount of lipoid has greatly varied. There is considerable evidence to show that the cortical cells of the adrenal gland, which are so largely developed in man as compared with the lower animals, are of great importance in the development of the body during its early period of existence, especially in respect to the development of the brain. It is also, as I have indicated previously, of great importance in relation to the development of the spermatozoa; and during pregnancy it increases in amount and provides doubtless a material necessary for the development of the growing embryo. The cholesterin esters are of extreme importance as constituents of the osmotic membranes of the blood corpuscles and the living cells of the body. The adrenal cortex was derived from a tissue which was once in close proximity to the early position of the sex glands in the abdomen. Adenoma of the cortex has been found associated with precocious development of the secondary sexual characters in males and females. But not only is there this mysterious correlation of the sex glands with the cortex of the adrenals, but there is also a correlation between the size of the brain and the development of the adrenal cortex in the animal series. The adrenal glands are invariably small in anencephalous monsters, as they are in brainless animals. The cortex of the adrenal is highly developed in the healthy human foetus; and all these facts support the argument that the cortical cells of the adrenal gland are a source of supply of the lecithin and cholesterin required for the enormous growth of the cerebral hemispheres in early life.

In the systematic investigation of the ductless glands which I have been making, in conjunction with Staff-Surgeon Kojima, I have been struck with the variability of the lipoid content, and one might have associated pathological conditions of the brain with its deficiency. Thus I recently made a post-mortem examination on a case of disseminated

sclerosis, and I was quite surprised to find the small amount of lipid present. Had I not known from Elliott's work that a few days' pneumonia would suffice to cause a great fall in the load of lipid, one might have been tempted to correlate a connexion between disseminated islands of nervous tissue, in which the myelin had disappeared, with a deficient functional activity of the adrenal cortex. With all the difficulties and pitfalls that confront one in the application of physiology to the study of the mind in health and disease, bio-chemistry, which is still in its infancy, seems to me to promise the most hopeful results.

Dr. J. G. SOUTAR proposed a vote of thanks to the President for his address and said: I desire to express my high appreciation of Dr. Mott's work in the domain of science he has dwelt upon this evening. Clinical alienists are greatly indebted to those who, like the President of our Section, are working to establish principles upon the basis of which progress can be made in the treatment of disease of the mind.

Sir GEORGE SAVAGE, in seconding the vote of thanks, said: It is not the custom to criticise the address of a President of Section, but it is usual to refer to it in proposing or seconding a vote of thanks. I have the greatest pleasure in seconding the vote of thanks, for I fully recognise the value of the contribution. In Dr. Mott we have not only the pathologist, but also the physiologist. The time has passed when the post-mortem room saw the beginning and end of pathology; the process of dying is a vital one. I was only this week struck with the living process of dying as seen by the shedding of leaves in the autumn. They have to be got rid of by a living process. This process is checked if the branch is injured while the tree is growing, and the dead leaves hang on through the winter. Death and pathology can only be understood by the man who thoroughly knows vital processes. Dr. Mott approaches his subject well prepared, and he has given us of the rich store he has. He has shown how dependent the brain is upon every other part of the body. Sir William Gull used to say the brain, like a gentleman, had many servants, but might be badly served by any one of them. The glands which were, up till recently, looked upon as quaint evidences of survival, and rather as relics than as actual agents, are now known to take influential parts in the growth and development of the body. The two great functions are to grow, and, as Dr. Mott has pointed out, to reproduce. The earliest years are spent chiefly on the first, then comes the critical time of life, and though some glands were very active even before birth, and still had important, though declining, functions during adolescence, other glands took on important reproductive functions represented by great mental and therefore brain changes. Dr. Mott has most clearly pointed out some of these relationships, and has indicated important lines of study. Calmly and clearly he has laid open to us the book of his life's work, and I can say that I am certain one and all of you will agree with me in according to him a hearty vote of thanks.

Section of Psychiatry.

April 28, 1915.¹

Dr. F. W. MOTT, F.R.S., President of the Section, in the Chair.

Cases of Hypothyroidism.

By ROBERT ARMSTRONG-JONES, M.D.

SCHIFF described cases of hypothyroidism in 1859, Gull in 1873, Ord in 1880, Kocher in 1883, and Dr. (now Sir) George H. Savage in 1890, the latter in association with mental disease. Dr. F. Mott took the subject of the ductless glands as the theme of his Presidential Address before this Section last year. The researches of Sir Victor Horsley have fully demonstrated the importance of the thyroid gland and the value of the secretion as a hormone in the physiology of metabolism. Halliburton states that a colloidal substance, a nucleo-albumin material, fills up the thyroid gland and that this substance, which has been demonstrated to contain a large percentage of iodine, increases with age. It has been suggested that the gland manufactures some thyro-antitoxin of the nature of an alkaloid, and it has been conclusively shown that this, through the medium of the blood, has a profound effect upon the organism. The removal of the gland is followed by great physical and mental disturbances, tremors at the rate of 500 per minute with marked tachycardia follow its overgrowth, whilst other physical and mental symptoms follow impairment of its functions or its removal. These latter symptoms are a notable overgrowth of the subcutaneous tissues, the body becomes heavy and clumsy, the hair thin and coarse, the skin rough and dry, and on the mental side there is listlessness, inactivity, apathy, and marked depression. That the thyroid gland exercises a marked influence upon the nutrition, circulation and heat-producing function is proved by the improvement in all these which results from administering the gland or its extract internally. The

¹ Clinico-Anatomical Meeting held at the Laboratory, Claybury Asylum.

action on the nervous system is no less striking. Absence of the gland in children gives rise to sporadic cretinism and in adults to myxœdema. The tremors that are noted in some mental cases with hypothyroidism are not unlike those found in certain cases of alcoholism and general paralysis, and it is possible for symptoms of hypothyroidism to appear when the gland itself is enlarged or cystic. The following cases are presented in relation to the subject.

The two following cases (of which photographs are shown) are of myxœdema with insanity, which recovered under the thyroid treatment.

CASE I.

T. B., was admitted into Claybury, March, 1913, having been transferred from the Essex County Asylum, Brentwood, where he had been for nine months. He was aged 59, married, an engineer's labourer. The history given was that his mental symptoms had been gradually coming on for nearly two years. On admission his mental state was of the depressed type, and he had delusions of persecution, voices talking to him through the wall. He complained of being watched and annoyed, and of having fumes blown upon him. He persisted that his "nature" had been taken from him, and he had some delusions of wealth, but he was generally apathetic, listless, and deficient in initiative. He spoke deliberately and the voice seemed husky. The heart sounds were weak, both heard, no murmurs. Pulse regular, and of good tension; no thyroid felt. Slight left facial paralysis, skin dry, hair stiff and wiry; urine acid, phosphates, 1020. Patient was treated with thyroid extract and soon showed marked improvement physically and mentally. He was discharged recovered after nine months' treatment.

CASE II.

W. N., aged 46, married. Admitted into Claybury, May, 1906. Before admission she had shown symptoms of insanity for three weeks, refusing food, and asking for poison. She had little sleep for over three months, and had lost flesh. She stated she was subject to and had "fainting fits"; she complained also that her hands and feet had been "swollen" for some years, and that she felt sensations of "pins and needles," in consequence of which the full normal use of her limbs was affected, but ordinary tactile sensation was unimpaired; she became strange, then confused, dull, and depressed, saying she felt her flesh was "rotten." On admission she suffered from melancholia with persecutory delusions, she could not work and had a feeling of inactivity, together with restlessness. Her face indicated myxœdema, eyes and face puffy, skin dry, hair rigid, the feet and hands were somewhat full and swollen, heart's action was feeble, but no murmur was heard, and she suffered from chronic dyspepsia. Pulse was regular, 76, and voice feeble, rather husky. She was treated with thyroid extract and she improved steadily and quickly

she began to employ herself usefully and was discharged, recovered, four months after admission. She was, however, readmitted a year later, stating she had suffered much privation and had home troubles. She had been unable to get the thyroid extract and to continue the treatment. She had developed visual and aural hallucinations with fear of harm, and had, at times, been violent. On admission she suffered from the same mental symptoms as before, and was unable to control or help herself. She was greatly worried and restless, with periods of listless apathy. She was again treated with thyroid extract, and became brighter and industrious. She was again discharged ten months after readmission.

The two following cases exhibited are still under treatment:—

CASE III.

C. A., widower, a labourer, aged 46. Admitted October, 1912. He was uneducated and unable to read or write, with visual and auditory hallucinations and mental confusion; there was some emotional depression and his expression was dull. He had a delayed mental reaction and was slow to answer questions, being confused and heedless of his surroundings. He was deficient in initiative. He felt in danger because he imagined there were two men who wanted to shoot him. His lips were blue and thick, pulse 80, heart sounds clear. Urine contained urates, no albumin. Face seemed puffy, skin harsh and dry, on hands, scalp and arms. He is generally dull, listless and apathetic, although occasionally irritable and quarrelsome. He reacted to thyroid extract and his pulse improved when he took $12\frac{1}{2}$ gr. a day, but tachycardia supervening, this was discontinued for a time and resumed later. He is improving under treatment. Family history records that his mother was an inmate of Tooting Bec Asylum.

CASE IV.

M. E. H., married, aged 43. Admitted December, 1912. Complained of headaches lasting six months; stated to have had diabetes twenty years ago. No children nor stillbirths; temperate in drink. Illness began with depression and low spirits owing to continued ill-health. Went to the asylum willingly. Slept a good deal before illness and would repeatedly ask the same questions. Good family history; mother living. Expression apathetic and listless on admission. Urine 1020, no albumin; anæmic. Nervous reflexes normal. On admission thyroid not felt. Takes no interest in things, generally dozing. Forgetful and cannot originate ideas. She has aural hallucinations, hears voices of people speaking and sounds in the head. Confused and wandering as to time and place, mistakes the identity of those about her. Three months after admission she became agitated and excited, hears her niece "May" calling, and she wanders about looking for her mother. Thyroid extract has been taken on and off since admission and there is some improvement.

Another case of hypothyroidism was that of a sporadic cretin.

CASE V.

S. D. Admitted into Horton, 1903, at the age of 49; is now aged 61 and looks younger owing to her short and stunted condition. Single, described as a servant. Her mental state was that of a generally slow person with impaired intelligence, voice somewhat husky and at times croaky. Is solitary, depressed and apathetic, and sits talking to imaginary voices. Tongue large and fills mouth in a bulky way. Skin somewhat dry and harsh. Had attacks of nose-bleeding when aged about 45. Can read and write and add simple numbers. Clean and tidy. Dislikes hearing piano played, turns her back to it. Pulse about 68 to 72. Stated to have menstruated irregularly, but periods stopped about her forty-ninth year. She has supraclavicular fatty fullnesses. Thyroid extract was administered for some time after admission, but with no apparent benefit, although the patient herself seemed to think she felt better whilst under its influence. It was discontinued owing to attacks of faintness which followed its use. She has only been in Claybury a few weeks and remains in the same condition as on her admission.

Another case resembling the Mongolian type is the following:—

CASE VI.

F. T., single, aged 25. Admitted November, 1912, suffering from congenital weak-mindedness with melancholia; stated to have been fairly quick at school in Eastbourne, to have had a fall, complaining of subsequent headache, also a "fit" six months before admission, to have had drinking bouts, taking only beer, and to have served three years in an inebriate reformatory. Father and mother married at about 20 years of age. Father intemperate before patient's birth. Three other children (a sister and two brothers) healthy. Urine 1035, urates. Thyroid not felt. Admitted hearing imaginary voices, depressed, and refusing food. She had suicidal tendencies. She is weak-minded and cannot add simple numbers correctly, and she cannot tell how many halfpence there are in a shilling. States she has been keeping bad company and drank and was convicted twenty-three times, that her father was a drunkard, and she was taken into a Salvation Army Home, and had been in an inebriate home for three years. Says two or three glasses of beer will cause her to be intoxicated. She has a mongoloid appearance, with rough, furfureous skin and dark dry hair.

Three other cases were exhibited of possible hypothyroidism, but with enlarged thyroid glands.

The Ductless Glands in 110 Cases of Insanity, with Special Reference to Hypothyroidism.

By Staff-Surgeon M. KOJIMA, I.J.N.¹

INTRODUCTION.

Part I: Source of material—Weight of ductless glands and tabulated statement—Comparative results and inferences—Conclusions regarding the weights of the ductless glands in the insane.

Part II: Microscopical investigation of the ductless glands and reproductive organs in hypothyroidism. Conclusions.

INTRODUCTION.

I have been engaged upon the investigation of the ductless glands in the insane at the suggestion and under the direction of Dr. Mott, F.R.S., &c., the Director of the Laboratory, to whom I express my sincere and deepest gratitude for the help and encouragement he has always so readily given me during my work. I am indebted to Dr. R. Armstrong-Jones for the clinical records of the cases, of which I shall give brief notes. The influence of the ductless glands upon bodily and mental functions in health and disease has formed the subject of a number of investigations, and much detailed information has been obtained, but our knowledge is still incomplete. There is a large amount of evidence to show that there is a functional correlation between the ductless glands and the nervous system, and the following investigation was undertaken to ascertain the extent of the changes to be found in the ductless glands of the insane. I intend first to give brief notes regarding the comparative weights of the glands in 110 consecutive autopsies, and follow with a description of four cases of hypothyroidism that occurred in the series.

I wish to express my best thanks to Messrs. Geary and Mann for their technical help throughout my work.

¹ From the Pathological Laboratory, Claybury Asylum.

PART I.

SOURCE OF MATERIAL.

Through the courtesy of Dr. Mott who made the post-mortem examinations, I have obtained all the material from 110 consecutive autopsies at Claybury Asylum. All the ductless glands were carefully removed from the body and freed from surrounding tissue, and carefully weighed. Those glands in which tumours or large cysts were found were not included in the series—e.g., those cases in which the ovaries were disintegrated owing to adhesive salpingitis, and the adrenals that could not be removed from the body without destruction. I was, generally speaking, fortunate enough, however, to obtain the material within a short time of death and without any signs of post-mortem change. To remove the external parathyroid I have taken special care to remove the organs of the throat entire, and then to dissect out the parathyroid according to the method of Erdheim, Getzowa, Rogers, and Ferguson.

WEIGHT OF DUCTLESS GLANDS AND TABULATED STATEMENT.

The weight of the thyroid gland in 110 male and female cases was found to vary considerably. The average weight in the male was 16.46 gm., and in the female 16.87 gm. The minimum weight in the male cases was 4.4 gm., and in the female cases was 4.5 gm. In both sexes between the ages of 30 and 50 there appears to be a comparative increase in the weight of the gland. The average weight in both male and female is extremely low, as compared with the minimum weight for the gland in other than mental cases given by various authors. In six male cases (12 per cent.) and eleven female cases (18 per cent.) the thyroid weighed less than 10 gm. (*see* Tables I to IV, pp. 24-33).

According to Ewald, v. Eiselsberg, Testut, Marine and Lenhart, and others, the normal minimum weight varies from 22 to 25 gm. According to Weibgen the gland in the male is larger than in the female, so that the mean weight of the gland in the female is 29.3 gm., whilst in the male it is 34.2 gm. It seems that in the asylum cases the average weight of the gland in the female is practically the same as that of the male. The maximum weight in the female is, generally speaking, greater than the maximum weight in the male, and there appears to be a much greater variation in the weights of the female cases than in the

male. The weight of the thyroid in proportion to the body-weight, according to Krause is 1 : 1,800 to 1,223, according to Huschke and Weibgen 1 : 1,800. The proportion in the insane is 1 : 3,232 in the male and 1 : 2,847 in the female.

The external parathyroid glands were carefully weighed in nineteen male cases and in twenty-two female cases — altogether in forty-one cases. The glands vary in number. In four male and six female cases four glands were found, each two on both sides, while in three male cases and in two female cases three glands were found, so that two glands occurred on one side of the lateral lobe of the thyroid and one on the other. In five male and three female cases only one gland was found, which in two male cases and one female case was found on the outside of the right lateral lobe of the thyroid gland, and in the remaining cases at the outside of the left lobe. In other cases of both sexes two glands were found at the outside of both lateral lobes of the thyroid gland. It is difficult to give an average weight, because the number of the glands in each case varies as above mentioned. The weight of each gland varies from 0.01 gm. to 0.07 gm. It is noteworthy that though in a case several glands were found, the weight of each gland does not show much difference. There is also no difference between glands of the right and the left sides. Yanase has found in 89 cases of children four glands in fifty cases, three in twenty-three cases, two in twelve cases, and one in four cases; Getzowa has also found four glands in one-third of 100 cases; v. Verebely in 108 out of 138 cases; Moller in two-thirds of 120 cases. I have found four glands in 21.9 per cent. of the cases. In a male with dementia præcox, aged 25, a double gland was found by microscopical examination. It is difficult to draw any conclusions respecting the relative weight of the thyroid and parathyroid glands.

The average weight of the pituitary body in the male is 0.56 gm., and in the female 0.65 gm. In the female, except those cases in which the large pituitary body has been found in conjunction with hypothyroidism, the average weight shows 0.62 gm. The smallest in the male cases is 0.22 gm., in the female cases 0.3 gm. The largest in the male is 0.9 gm., and in the female 1.2 gm., and the next one is 1.05 gm. In a female with general paralysis, aged 37, who died after a few days' illness from broncho-pneumonia after giving birth to a child, the anterior part of the pituitary body was enlarged, and weighed 0.7 gm., while the pineal gland weighed 0.15 gm., and the thyroid 14.10 gm. (*vide* Tables I to IV, pp. 24-33).

TABLE I.—FIFTY MALE CASES.

N. B.—Body weight in kilogrammes, the other organs in grammes.

No.	Name	Age on admission	Age at death	Disease	Cause of death and complications	WEIGHT									
						Body	Brain	Pituitary body	Pineal gland	Thyroid	Adrenals		Testicles		
											Right	Left	Right	Left	
1	E. J. A.	21	21	Insanity of adolescence	Pulmonary tuberculosis	57.0	1,370	0.55	0.47	18.1	6.55	5.52	10.20	11.10	
2	F. F.	25	25	General paralysis	Exhaustion of general paralysis, broncho-pneumonia	34.0	1,200	0.58	0.18	11.70	6.20	4.20	6.52	6.42	
3	*E. S.	24	25	Dementia praecox	Pneumonia	46.0	1,330	0.57	0.17	18.3	8.8	10.70	21.2	16.1	
4	W. L.	25	26	Adolescent melancholia	Acute dysentery, commencing pneumonia	59.0	1,360	0.60	0.10	20.1	7.80	8.90	11.00	9.89	
5	E. E. H.	28	29	Congenital imbecility	Pulmonary tuberculosis, commencing tuberculosis of intestine	43.0	1,315	0.60	0.15	16.0	6.50	3.00	10.30	9.90	
6	H. H.	27	34	Secondary (terminal) dementia	Pulmonary tuberculosis	51.0	1,525	0.67	0.12	23.30	8.00	8.45	10.2	10.45	
7	G. W. P.	27	34	Mania	Acute dysentery, broncho-pneumonia	59.0	1,480	0.60	0.12	10.72	7.72	6.62	10.8	11.20	
8	J. T.	27	34	Manic depressive insanity	Pulmonary tuberculosis, mixed infection, gangrene of lung, pneumonia	61.0	1,325	0.73	0.03	21.63	6.83	6.8	7.6	9.50	
9	A. E. L.	33	35	General paralysis	General paralysis, broncho-pneumonia	55.5	1,425	0.80	0.25	13.10	6.40	5.4	21.2	17.90	
10	A. C.	36	37	General paralysis	Advanced general paralysis, broncho-pneumonia with cavitation and commencing gangrene	—	1,150	0.60	0.10	26.55	10.15	7.35	13.8	13.90	
11	G. H. S.	29	37	Epileptic dementia	Pleurisy, cardiac failure, fatty degeneration of heart, hypostatic congestion of both lungs	63.0	1,515	0.75	0.25	14.00	6.50	7.50	11.5	13.50	

12	S. P.	35	37	Melancholia	51.0	1,460	0.65	0.15	32.25	5.55	6.1	10.1	9.25
13	F. T.	37	39	Recurrent melancholia	40.0	1,230	0.60	0.17	9.52	8.22	7.02	10.77	11.48
14	P. H. B.	38	39	Tabo-paralysis	46.0	1,175	0.34	0.05	6.72	5.72	8.22	8.2	9.30
15	C. W. S.	39	40	General paralysis	58.0	1,460	0.47	0.20	19.00	5.90	5.50	12.5	13.5
16	W. F. C.	40	41	Tabo-paralysis	—	1,410	0.67	0.17	12.60	13.50	5.40	12.40	11.90
17	C. A.	40	41	Epileptic dementia	53.0	1,370	0.50	0.20	30.50	5.20	4.20	10.10	9.80
18	J. C.	24	41	Chronic mania	57.0	1,490	0.90	0.15	11.60	6.40	5.70	10.20	9.80
19	H. A. R.	41	42	Epileptic dementia	56.0	1,285	0.22	0.12	15.17	6.87	6.17	13.17	11.47
20	C. K.	41	42	General paralysis	47.0	1,488	0.83	—	15.10	8.30	9.10	18.30	17.50
21	C. J. G.	41	43	General paralysis	54.5	1,370	0.80	0.25	18.00	5.50	6.50	14.50	12.60
22	C. W. S.	45	46	General paralysis	56.0	1,240	0.40	0.20	30.00	8.20	7.80	12.00	12.50
23	W. S.	47	47	Acute mania	57.0	1,345	0.55	0.13	16.90	2.90	3.80	12.30	11.90
24	W. H. T.	46	48	Recurrent mania	71.0	1,610	0.50	0.20	13.25	4.25	3.42	6.25	4.42
25	H. L.	48	49	General paralysis	51.0	1,500	0.70	0.50	15.9	5.5	4.7	24.4	23.2
26	C. B.	50	50	Melancholia	43.0	1,345	0.45	0.15	11.40	8.02	6.52	10.32	12.22

* A persistent thymus was found in this case.

TABLE I—(continued).

No.	Name	Age on admission	Age at death	Disease	Cause of death and complications	WEIGHT								
						Body	Brain	Pituitary body	Pineal gland	Thyroid	Adrenals		Testicles	
											Right	Left	Right	Left
27	C. B. P.	49	50	General paralysis	Dilated aorta, bronchitis, emphysema	70.0	1,200	0.70	0.20	17.00	6.95	7.45	16.45	18.45
28	W. D.	46	51	General paralysis	Pneumonia, morbus cordis	57.0	1,245	0.70	0.10	22.30	6.10	6.50	10.92	12.50
29	R. J. Y.	31	51	Congenital imbecility	Pneumonia, stone in kidney	34.0	1,085	0.50	0.10	4.4	3.05	3.5	4.30	4.10
30	W. G.	53	54	Katatonic ecstasy of primary dementia	Obsolescent tubercle, chronic renal disease, bronchitis, emphysema, arterio-sclerosis, fatty degeneration of the heart, internal hydrocephalus	52.5	1,580	0.61	0.31	6.6	3.81	3.51	10.3	10.7
31	R. P. F.	54	55	Confusional insanity	Hæmorrhagic pancreatitis	58.0	1,350	0.60	—	11.7	9.9	5.6	12.5	11.2
32	W. C. W.	51	55	Melancholia	Pulmonary tuberculosis, heart failure, tubercular ulceration of intestine	43.0	1,250	0.38	0.10	8.90	3.90	2.80	—	6.10
33	J. Y.	48	57	Insanity with epilepsy	Epilepsy, heart failure	—	1,430	0.67	0.07	32.17	8.5	7.7	19.7	18.7
34	T. S.	52	59	Insanity with epilepsy	Broncho - pneumonia, gangrene of foot, heart failure	41.0	1,515	0.70	0.25	15.50	6.50	6.80	9.80	10.90
35	J. J.	55	58	Delusional insanity	Secondary carcinoma affecting nearly all the organs, especially the lungs	51.0	1,335	0.4	0.1	13.5	12.5	9.5	18.5	19.5
36	J. E. T.	56	59	Acute mania	Exhaustion of general paralysis, bronchitis, emphysema, fatty heart	51.0	1,255	0.55	0.25	30.6	6.2	6.5	11.0	10.1
37	F. L.	58	60	Senile melancholia	Cardiac failure, broncho-pneumonia, bronchitis, emphysema, arterio-sclerosis, generalised, including cerebral arteries	62.0	1,315	0.58	0.25	18.5	5.8	6.9	10.5	11.3

38	G. W. B.	56	60	Subacute mania	Exhaustion of senile dementia, bronchitis, emphysema, heart failure	46.5	1,285	0.48	0.15	12.7	3.7	3.4	12.8	10.7
39	J. A. W.	59	60	General paralysis	Purulent bronchitis, atrophic emphysema, fatty heart	54.0	1,345	0.62	0.07	20.6	4.1	4.2	10.1	7.2
40	H. M.	60	62	General paralysis	General paralysis, broncho-pneumonia	—	1,625	0.55	0.25	14.55	6.75	4.1	24.75	27.55
41	J. W. M.	60	62	General paralysis	Gangrene of lung, arterio-sclerosis, general paralysis, renal disease	61.0	1,270	0.45	0.2	27.7	11.5	9.3	11.5	16.5
42	J. M.	58	63	Recurrent melancholia, epilepsy	Cancer of cesophagus, broncho - pneumonia, chronic Bright's disease, chronic cystitis	73.0	1,585	0.46	0.23	12.6	5.9	5.6	14.45	15.9
43	A. H.	57	63	Dementia with epilepsy	Bronchitis, emphysema, arterio - sclerosis, advanced chronic Bright's disease, asphyxia owing to obstruction of the glottis, opening into the trachea by a clot of mucus and blood	59.0	1,430	0.45	0.15	11.55	5.25	4.45	9.0	8.1
44	W. R.	63	63	Senile general paralysis	Bronchitis, broncho-pneumonia, arterio-sclerosis, adenoma of right adrenal	68.5	1,420	0.6	0.15	21.00	—	17.5	14.5	18.0
45	W. C. S.	63	64	Dementia	Pneumonia, heart failure, renal arterio-sclerosis	61.0	1,215	0.62	0.055	10.6	8.02	7.1	12.2	10.8
46	W. T.	50	65	Alcoholic dementia	Pneumonia, arterio-sclerosis	60.0	1,265	0.5	0.1	8.65	3.55	4.25	10.3	12.35
47	E. O.	66	66	Dementia with epilepsy	Broncho - pneumonia, aortic dilatation	57.0	1,270	0.47	0.20	13.5	6.28	7.02	15.92	14.3
48	T. C.	48	69	Mania with epilepsy	Pneumonia, chronic Bright's disease	59.0	1,530	0.65	0.27	16.6	8.7	6.95	11.3	10.9
49	E. D.	68	70	Acute melancholia	Fatty heart, broncho-pneumonia, calculous pyelitis	61.0	1,360	0.5	0.16	11.7	5.9	7.9	14.8	14.2
50	R. W.	70	79	Senile melancholia	Pulmonary tuberculosis, pneumonia	69.0	1,130	0.68	0.08	17.18	6.99	6.53	14.47	13.7

TABLE II.—SIXTY FEMALE CASES.

No.	Name	Age on admission	Age at death	Disease	Cause of death and complications	WEIGHT									
						Body	Brain	Pituitary body	Pineal gland	Thyroid	Adrenals		Ovaries		
											Right	Left	Right	Left	
1	E. E.	19	19	Mental confusion	Disseminated sclerosis, broncho-pneumonia	51.0	1,230	0.62	0.25	20.50	3.00	3.70	4.20	5.90	
2	E. K.	22	25	Insanity of adolescence	Pulmonary tuberculosis, tubercular deposits in liver, spleen, and intestine, caseous mesenteric glands	—	1,080	0.60	0.25	20.06	8.30	9.30	2.95	1.60	
3	A. H.	25	26	Primary dementia	Acute pulmonary tuberculosis, tubercular ulceration of intestine, aortic hypoplasia	48.0	1,175	0.60	0.10	9.80	3.20	2.80	2.90	2.40	
4	P. A. C.	26	27	Congenital mental weakness	Septicæmia, gangrene of lung, bronchiectasis, hydrocephalus	51.0	—	0.80	—	11.50	3.50	4.70	4.23	5.02	
5	F. C.	27	28	Agitated melancholia	Miliary tuberculosis pulmonalis, subacute dysentery	34.0	1,175	0.45	0.05	27.85	6.05	6.85	3.05	1.75	
6	N. D.	31	33	Dementia præcox	Broncho-pneumonia	56.0	1,220	0.40	0.15	7.70	2.80	3.40	1.80	2.70	
7	M. A.	31	33	Congenital mental weakness	Pulmonary tuberculosis, fatty liver	—	—	0.60	0.10	14.00	4.70	4.20	2.30	2.00	
8	F. L. A.	24	34	Adolescent mania	Pulmonary tuberculosis of comparative recent origin, dysentery, fatty liver, hour-glass stomach	34.0	1,110	0.80	0.16	22.03	8.95	4.60	—	—	
9	M. C.	31	35	Acute mania	Asphyxia, hæmorrhage into the bronchi of both lungs, grave anæmia	53.0	1,270	0.89	0.35	51.20	5.25	4.90	4.90	8.15	
10	E. S. M.	37	37	General paralysis	Broncho - pneumonia, general paralysis	43.0	1,130	0.70	0.15	14.10	7.10	5.70	3.90	2.51	

11	A. F.	32	37	Mania, post-influenza Melancholia	Morbus cordis, chronic Bright's disease Pulmonary tuberculosis	61.0	1,230	0.62	0.27	15.95	8.40	8.10	—	9.50
12	M. E.	30	37	Melancholia	Pulmonary tuberculosis	39.0	1,145	0.80	0.19	11.90	4.90	3.20	4.20	4.00
13	E. S.	33	39	Dementia præcox	Pulmonary tuberculosis	42.0	1,145	0.60	0.22	13.70	4.15	1.85	3.75	1.85
14	S. S.	19	39	Recurrent mania	Broncho - pneumonia, grave anæmia	49.0	1,130	0.85	0.23	13.50	7.90	7.40	4.60	7.90
15	M. S.	35	40	Recurrent mania	Pulmonary tuberculosis, pleurisy with effusion	39.0	1,275	0.75	0.18	52.52	5.00	6.20	3.85	5.40
16	F. M. H.	39	40	Melancholia	Bronchitis, broncho- pneumonia, commencing chronic dysentery	38.5	1,275	0.45	0.10	11.40	6.20	6.80	4.50	4.30
17	B. H.	41	41	General paralysis	Broncho - pneumonia, general paralysis, plaques of syphilitic aortitis	34.5	1,035	0.73	0.12	11.50	4.15	3.87	3.41	2.82
18	S. G.	32	43	Epileptic melancholia	Broncho-pneumonia	40.5	1,275	0.76	0.13	12.48	3.16	2.25	4.32	3.85
19	E. S. H.	41	43	Subacute mania	Dysentery, old salping- itis with adhesion both sides	46.0	1,190	0.48	0.23	22.10	5.15	6.75	—	—
20	J. I. E. B.	42	43	General paralysis	Bronchitis, emphysema, general paralysis	37.0	1,045	0.58	0.15	10.10	6.00	5.25	1.50	3.10
21	A. R.	35	43	Melancholia	Bronchitis, broncho- pneumonia, brown pig- mentation of heart, symmetrical gangrene of toes (great and second)	42.0	—	0.75	0.15	30.30	6.70	4.00	3.00	1.95
22	L. M.	22	43	Mania with epilepsy	Capillary bronchitis, broncho - pneumonia, heart failure	51.0	1,240	0.80	0.18	16.70	7.80	7.50	3.10	3.60
23	A. R.	43	43	Epileptic dementia	Cerebral tumour, hypo- static congestion of the lungs	47.5	1,570	0.85	—	21.65	7.65	5.95	3.45	4.65
24	E. T. L.	36	44	Melancholia	Pulmonary tuberculosis, pericarditis, chronic dysentery	27.0	1,315	0.40	0.10	13.10	3.90	3.75	3.80	3.90
25	E. M. E.	44	44	Confusional insanity (alcoholic?)	Broncho - pneumonia, hypothyroidism, old parametritis, grave anæmia	43.0	1,080	0.77	0.12	9.2	4.25	4.55	1.20	1.60

TABLE II—(continued).

No.	Name	Age on admission	Age at death	Disease	Cause of death and complications	WEIGHT								
						Body	Brain	Pituitary body	Pineal gland	Thyroid	Adrenals		Ovaries	
											Right	Left	Right	Left
26	M. A. C.	44	46	General paralysis	Bronchitis, broncho-pneumonia, general paralysis	49.0	945	0.30	0.11	12.69	4.22	3.82	3.70	6.20
27	A. B.	46	46	Confusional insanity	Broncho - pneumonia, cardiac failure, hæmorrhagic pancreatitis	42.0	1,165	0.48	0.15	10.50	6.40	6.50	0.80	0.77
28	M. T. A.	47	47	Confusional insanity	Edematous glottis, bronchitis, emphysema, hypothyroidism, myxœdema, chronic Bright's disease	78.0	1,215	1.05	0.25	33.60	5.08	5.25	3.35	—
29	E. C.	46	48	Mania	Lobar pneumonia, chronic parenchymatous nephritis	53.0	1,240	0.77	0.07	9.27	7.77	13.57	1.70	1.70
30	N. S.	39	49	Epileptic mania	Subacute dysentery, bronchitis, emphysema, fatty heart, disorganised suprarenals, its cortex and medulla both affected	55.5	1,295	0.60	0.10	27.30	16.20	10.30	2.40	2.80
31	E. D.	48	49	General paralysis	General paralysis, bronchitis, broncho-pneumonia	66.0	1,085	0.55	0.20	11.10	5.45	6.40	1.55	1.05
32	G. P.	50	51	Confusional insanity	Broncho - pneumonia, hypothyroidism	51.0	1,055	0.48	0.06	7.49	3.98	4.73	1.75	1.55
33	E. G.	51	53	Manic depressive insanity	Exhaustion of chronic dysentery	46.0	1,145	0.75	0.30	26.50	7.20	6.70	2.35	1.95
34	H. B.	53	53	Alcoholic dementia	Morbus cordis, arteriosclerosis	61.0	1,180	0.70	0.30	8.70	7.80	5.00	3.70	—
35	E. H.	42	57	Climacteric melancholia	Pulmonary tuberculosis, tuberculous ulceration of intestine, gangrene of lung	24.0	1,225	0.40	0.12	10.97	2.86	3.19	3.80	4.00
36	E. P.	56	58	General paralysis	Bronchitis, emphysema, hypostatic pneumonia, commencing cerebral tumour, general paralysis	61.0	1,020	0.75	0.22	8.25	8.72	5.90	1.45	—

37	A. M. S.	48	58	Dementia	Pulmonary tuberculosis	49.0	1,160	0.65	0.25	23.75	9.20	3.75	2.50	2.70
38	H. R.	39	59	Recurrent mania	Cardiac failure from fatty heart and coronary sclerosis, arterial sclerosis, renal sclerosis	60.5	1,090	0.61	—	13.15	4.10	2.72	2.85	3.12
39	E. B.	57	59	Presenile dementia	Purulent bronchitis, pneumonia	39.0	1,105	0.57	0.09	12.85	4.80	4.50	1.90	1.95
40	E. A. W.	60	60	Acute mania	Broncho - pneumonia, heart failure	41.0	1,280	0.70	0.20	23.80	6.60	6.00	2.60	2.55
41	M. A. D.	44	61	Dementia	Perforation from stercoraceous ulceration just above sigmoid flexura, arterio-sclerosis	57.0	1,265	0.83	0.2	16.15	5.16	4.50	2.40	2.20
42	R. A. L.	62	62	Dementia	Cardiac failure, bronchitis, pleurisy, cirrhosis of liver, granular contracted kidneys, pachymeningitis	88.0	1,130	0.83	0.07	22.00	5.10	4.80	1.35	1.35
43	E. A. H.	44	63	Mania	Bronchitis, emphysema, chronic Bright's disease	40.0	1,140	0.80	0.10	13.00	5.00	6.20	2.50	1.65
44	C. H.	43	65	Dementia	New growth of sigmoid flexures with gangrenous ulceration, perforation, peritonitis	38.0	1,000	0.70	0.10	6.90	5.70	5.20	—	—
45	A. J.	57	67	Melancholia (alcoholic?)	Acute bronchitis, emphysema, chronic Bright's disease, hypothyroidism, old adhesive salpingitis	57.0	1,160	1.20	0.07	12.60	4.95	4.65	1.40	—
46	E. N.	47	67	Melancholia	Heart failure, chronic Bright's disease, general mechanical congestion, cyst of Broca's convolution, general anasarca, ascites, recent pulmonary infarction, hydrothorax	74.0	1,335	0.40	0.28	12.50	3.10	2.80	4.20	3.80
47	E. G.	68	68	Senile melancholia	Purulent bronchitis, emphysema	37.0	1,265	0.65	0.42	22.25	5.95	5.85	1.65	—
48	E. W.	54	69	Melancholia	Hæmorrhagic pancreatitis, advanced arteriosclerosis of all organs, including brain, gastric hæmorrhage, advanced contracted granular kidneys	46.0	1,115	0.70	0.20	4.50	7.00	5.40	2.00	1.70

TABLE II—(continued).

No.	Name	Age on admission	Age at death	Disease	Cause of death and complications	WEIGHT								
						Body	Brain	Pituitary body	Pineal gland	Thyroid	Adrenals		Ovaries	
											Right	Left	Right	Left
49	E. H.	49	69	General paralysis	Purulent bronchitis, fatty heart, chronic Bright's disease	73.0	1,025	0.72	0.17	36.27	—	—	—	—
50	A. A.	49	70	Chronic mania	Pneumonia, pneumococcal meningitis	53.0	1,285	0.90	—	16.8	6.50	7.20	2.10	1.40
51	A. H.	70	72	Senile dementia	Cardiac failure	54.0	1,035	0.75	0.15	9.50	3.50	4.10	1.30	1.10
52	A. A.	72	74	Senile dementia	Bronchitis, bronchopneumonia, pachymeningitis hæmorrhagica	57.0	1,285	0.75	0.25	10.75	7.25	5.65	2.55	6.25
53	A. H.	71	75	Dementia secondary to melancholia	Arterio-sclerosis, chronic Bright's disease, atrophic emphysema	59.0	1,315	0.73	0.28	27.90	7.80	6.50	1.10	1.99
54	E. R.	73	75	Hypochondriacal melancholia	Subacute dysentery, fatty heart, bronchitis, emphysema, senile decay	43.0	1,205	0.60	0.20	9.50	4.60	5.10	0.80	0.81
55	J. B.	76	78	Senile mania	Subacute dysentery, senile decay, bronchitis	51.5	1,080	0.39	0.29	22.75	5.99	6.19	1.29	1.89
56	E. E. W.	60	81	Melancholia	Broncho - pneumonia, senile decay, advanced arterio-sclerosis	38.0	1,160	0.63	0.21	12.32	3.24	4.12	3.23	2.25
57	M. C.	65	82	Senile melancholia	Bronchitis, emphysema, heart failure, senile decay, granular contracted kidney	44.0	1,150	0.70	0.57	10.40	3.95	4.10	2.20	1.90
58	E. P.	79	83	Senile dementia	Broncho - pneumonia, senile decay	41.0	1,245	0.80	0.25	12.70	4.25	4.15	2.00	2.20
59	S. A. T.	77	84	Dementia	Subacute dysentery, senile decay, arterio-sclerosis	53.0	1,010	0.40	0.16	10.40	8.20	5.30	1.00	1.70
60	E. B.	76	88	Chronic mania	Senile decay, bronchopneumonia	34.0	—	—	0.30	18.40	7.65	3.60	2.10	1.65

TABLE III.—AVERAGE WEIGHT OF FIFTY MALE CASES.

Age-period	THYROID			Pituitary body	Pineal gland	ADRENALS		TESTICLES		Body	Brain
	Maximum	Minimum	Average			Right	Left	Right	Left		
20-29 (5 cases)	20.1	11.7	16.84	0.584	0.214	7.17	6.464	11.844	10.64	47.8	1315.0
30-39 (9 cases)	35.25	6.72	17.86	0.637	0.137	7.23	7.38	11.57	10.83	53.3	1365.0
40-49 (11 cases)	30.50	11.60	18.91	0.594	0.214	6.59	5.66	13.28	11.69	56.05	1415.2
50-59 (11 cases)	32.17	4.4	15.82	0.569	0.163	6.85	6.034	12.379	13.5	50.05	1327.09
60-69 (12 cases)	27.7	8.65	13.77	0.448	0.143	5.35	6.03	10.105	13.63	53.7	1138.7
70-79 (2 cases)	17.18	11.7	14.44	0.59	0.12	6.44	7.21	14.63	13.9	65.0	1245.0
20-70 (50 cases)	35.25	4.4	16.46	0.56	0.167	6.551	6.22	11.91	12.91	53.2	1303.6

TABLE IV.—AVERAGE WEIGHT OF SIXTY FEMALE CASES.

Age-period	THYROID			Pituitary body	Pineal gland	ADRENALS		OVARIES		Body	Brain
	Maximum	Minimum	Average			Right	Left	Right	Left		
19 (1 case)	—	—	20.50	0.62	0.25	3.00	3.7	4.2	5.9	51.0	1230.0
20-29 (4 cases)	27.85	9.8	17.3	0.61	0.13	5.26	5.91	2.37	2.69	44.3	1143.3
30-39 (9 cases)	51.20	7.7	18.23	0.69	0.2	6.01	4.7	3.63	4.8	47.1	1172.5
40-49 (17 cases)	52.52	9.2	18.55	0.59	0.14	6.14	5.98	3.81	3.17	46.4	1202.8
50-59 (8 cases)	26.5	7.49	13.95	0.61	0.19	6.08	4.56	2.53	2.54	48.9	1116.0
60-69 (10 cases)	36.27	4.50	16.99	0.753	0.171	4.856	4.49	1.81	1.325	54.6	1171.5
70-79 (6 cases)	22.75	9.5	16.2	0.68	0.23	5.94	5.79	1.52	2.24	52.9	1200.8
80-89 (5 cases)	18.4	10.4	12.84	0.63	0.39	5.45	4.25	2.1	1.94	42.0	1141.2
19-89 (60 cases)	52.52	4.5	16.87	0.652	0.198	5.81	5.2	2.62	2.97	48.04	1173.2

According to Boyce and Beadles, the average weight is 0.453 gm. in the male and 0.6 gm. in the female. These observers investigated the weights in fifty cases of each sex dying in Colney Hatch Asylum. According to Schonemann, the weight of the pituitary body increases from birth to 10 years of age. At 20 years of age it is 0.54 gm., at 30, 0.8 gm., and from 20 to 30, 0.63 gm., while at the age of 50 it is 0.6 gm. Benda gave the weight as 0.5 to 1.0 gm., and he has concluded that under and over these limits it may be considered as evidence of hypo- or hyper-plasia associated with histological changes. An investigation carried out by Dr. Candler at Dr. Mott's suggestion gave the average weight as 0.469 gm. in eighteen males, and 0.567 gm. in twenty-six females. As Boyce and Beadles have pointed out, it seems that the average weight in the male is, in general, less than in the female, but there are, of course, many exceptional cases.

The average weight of the pineal gland is 0.167 gm. in the male and 0.198 gm. in the female. There is no difference corresponding to any particular disease. All the post-mortems were conducted on adults over 20 years of age, and therefore the variations in weight do not come into account. The largest gland found in the male was 0.5 gm., and in the female 0.57 gm. The smallest in the male was 0.03 gm., and in the female 0.05 gm. In a female with chronic mania, aged 70, the gland was absent.

The weight and condition of the adrenals depend upon many factors—for example, the cause of death, the duration of illness causing death, and the time elapsing before the post-mortem is made—that it is difficult to draw any conclusions regarding the comparative weight of these organs, but it is interesting to note that the weight of the right gland in thirty-eight cases of sixty females was heavier than the left. Elliott gives the weight of an adult male as 4 gm. to 5 gm. Testut gives 6 gm. to 7 gm. as the average weight of each adrenal. According to v. Neusser and Wiesel the weight is 11.6 gm. in the male and 10.6 gm. in the female. Parhon and Zugraw showed that it is less heavy in the female sex in the insane. My cases also show that the average weight in the female is less than in the male. There is no definite correlation between body-weight and weight of adrenal glands.

It is very difficult to draw conclusions regarding the weight of the testicles and the ovaries. Generally speaking, the ovaries reach a fairly heavy weight between the age-period 30 to 50 years. But in two cases of females, aged 28 and 25, the ovaries were small—e.g., the left ovaries were 1.75 gm. and 1.6 gm., while the right were 3.05 gm.

and 2.95 gm. respectively. In a case of dementia præcox, aged 33, who died after a few days' illness from broncho-pneumonia, the ovaries were also small, so that the right ovary was 1.8 gm., while the left was 2.7 gm. The weight of the testicles showed no difference according to the age. The weight varies considerably, so that the weight of the right testicle varies from 4.3 gm. to 24.75 gm. and that of the left from 4.1 gm. to 27.55 gm. In certain cases the testicles were comparatively small.

Case	Age	Right testicle	Left testicle
General paralysis ...	25 ...	6.52	6.42
Tabo-paralysis ...	39 ...	8.2	9.3
Manic depressive insanity	34 ...	7.6	9.5
Recurrent mania ...	48 ...	6.25	4.42
Melancholia ...	55 ...	Castrated for tubercular epididymitis two years before onset	

COMPARATIVE RESULTS AND INFLUENCE.

It may be inferred from the above statement that the thyroid gland in both male and female in the insane is generally smaller than the normal, but it will be noted that there are considerable variations in the weights of the thyroid glands, especially is this noticeable among the female cases that died just about the climacterium or before or after that period of life.

MALES WITH HYPOTHYROIDISM.

In six male cases and in eleven female cases the weight of the thyroid gland was under 10 gm. In a male patient with congenital imbecility, aged 51, all the glands were small, the thyroid weighing 4.4 gm. only. In certain male cases a correlation seems to exist between the weights of the glands, so that all the glands are small.

Case	Age	Pituitary body	Pineal gland	Thyroid	Suprarenals		Testicles	
					Right	Left	Right	Left
Tabo-paralysis...	39	0.34	0.05	6.72	5.72	8.22	8.2	9.3
Congenital imbecility ...	51	0.5	0.1	4.4	3.05	3.5	4.3	4.1
Melancholia ...	55	0.38	0.1	8.9	3.9	2.8	—	6.1
Katatonic ecstasy of dementia præcox ...	54	0.61	0.31	6.6	3.81	3.51	10.3	10.7
Recurrent mania ...	39	0.6	0.17	9.52	8.22	7.02	10.77	11.48

There are many conditions which need not be detailed that may exist to account for such differences of weight as occur; consequently it is not permissible with so few cases to draw any conclusions from the above.

FEMALES WITH HYPOTHYROIDISM.

In certain female cases the thyroid gland and the ovaries were small; on the contrary, the pituitary body and the pineal gland were large. It seems, therefore, that there is a functional correlation between these glands:—

Case	Age	Pituitary body	Pineal gland	Thyroid	Suprarenals		Ovaries	
					Right	Left	Right	Left
Primary dementia ...	26	0·6	0·1	9·8	3·2	2·8	2·9	2·4
Confusional insanity ...	44	0·77	0·12	9·2	4·25	4·55	1·2	1·6
Mania ...	48	0·77	0·07	9·27	7·77	13·57	1·7	1·7
Post-hemiplegic dementia (general paralysis) ...	58	0·75	0·22	8·25	8·72	5·90	1·45	—
Alcoholic dementia ...	53	0·7	0·30	8·70	7·80	5·00	3·70	—
Senile dementia ...	72	0·75	0·15	9·5	3·5	4·1	1·3	1·1
Hypochondriacal melan- cholia ...	75	0·6	0·2	9·5	4·6	5·1	0·8	0·81

In the subjoined two female cases, which I will describe later as hypothyroidism, the pituitary bodies were very large.

Case	Age	Pituitary body	Pineal gland	Thyroid	Suprarenals		Ovaries	
					Right	Left	Right	Left
Confusional insanity ...	47	1·05	0·25	33·6	5·08	5·25	3·35	Cystic
Melancholia (alcoholic?)	67	1·20	0·07	12·6	4·95	4·65	1·4	—

Parhon showed that the average weight of the thyroid of the insane is greatest in the affective psychoses—i.e., mania, melancholia, mania depressiva—and least in epilepsy. In my cases there is no definite relation between the weight and the affective psychosis in the male. In thirteen female cases the thyroid glands were over 20 gm. These cases are as follows: Adolescent mania, one case; insanity of adolescence, one case; melancholia, three cases; mania, five cases; dementia, two cases; confusional insanity, one case. It seems difficult to confirm the conclusion of Parhon, but it is noteworthy that the thyroid gland in many female cases of the affective psychoses is large.

In the subjoined female case of dementia præcox, aged 33, who died from broncho-pneumonia after a few days' illness, all the glands were comparatively smaller than the average.

Case	Age	Pituitary body	Pineal gland	Thyroid	Suprarenals		Ovaries	
					Right	Left	Right	Left
Dementia præcox ...	33	0·4	0·15	7·7	2·8	3·4	1·8	2·7

The weight of the pituitary gland has no definite relation to the nutrition of the body, to the form and the duration of the insanity, or to

the nature of the other organic diseases which were met with at the autopsy, except in the cases of hypothyroidism.

The differences in weight of the glands previously described seem to indicate that there may be an interference with the normal metabolism of the ductless glands in the cases recorded. It is possible, therefore, that the mental condition may in some way be associated more or less with a deranged function of the ductless glands interfering with the normal bio-chemical equilibrium of the hormones.

CONCLUSIONS REGARDING THE WEIGHT OF THE DUCTLESS GLANDS IN THE INSANE.

(1) The average weight of the thyroid gland in both male and female subjects of insanity is generally smaller than the normal. Thus the average weight is 16.46 gm. in the male and 16.87 gm. in the female; in 12 per cent. of the male and 18 per cent. of the female the thyroid glands were under 10 gm. There are considerable variations in the weight, especially among the female cases at the climacterium or shortly before and after that period. In many female cases of affective psychosis the thyroid gland was very large.

(2) The weights of the external parathyroid glands vary from 0.01 gm. to 0.07 gm. In 21.9 per cent. of the cases four glands were found.

(3) The average weight of the pituitary body is 0.56 gm. in the male and 0.62 gm. in the female. In the female cases in which the thyroid glands were small the pituitary body was generally large. The weight has no definite relation to the nutrition of the body, to the form and the duration of the insanity, or to the nature of the other organic diseases, except in certain cases of hypothyroidism.

(4) The average weight of the pineal gland of the adult is 0.167 gm. in the male and 0.198 gm. in the female. There is no difference corresponding to any particular disease.

(5) The adrenals of the male are, generally speaking, heavier than in the female. There is no definite correlation between body-weight and weight of these glands.

(6) No definite conclusion can be arrived at regarding the weight of the reproductive glands in the male and female in relation to body-weight or mental disease, but in certain female cases, in which the thyroid gland was small, the ovaries were also remarkably small.

PART II.

THE HISTOLOGICAL EXAMINATION OF THE THYROID GLAND AND OTHER DUCTLESS GLANDS.

As already mentioned, in six male and in eleven female cases the thyroid glands were under 10 gm. In two cases out of the above eleven females marked fibrotic changes of the glands were seen by microscopic examination. Moreover, in two other female cases of confusional insanity and of melancholia (alcoholic?) the thyroid glands were 33.6 gm. and 12.6 gm. respectively. Microscopic examination exhibited a marked increase of the fibrous tissue and an atrophy of the gland structure. These two cases may also be regarded as hypo-function of the thyroid—viz., hypothyroidism. As above mentioned, in four female cases of hypothyroidism it was found that the thyroid glands showed evidence of morbid changes under the microscope. In three cases, in addition to the fibrous hyperplasia there was a marked lymphoid infiltration. This subject of hypothyroidism will now be dealt with, combined with a description of the other ductless glands.

THE MATERIAL.

All the materials were preserved in formalin—Müller's, Flemming's, or Zenker's fluid. Some preparations from the thyroid gland embedded in celloidin were stained with hæmatoxylin-eosin and by Van Gieson's method. The preparations from other glands, embedded in paraffin or celloidin, were also stained with hæmatoxylin-eosin, Van Gieson's and Heidenhain's eosin staining fluids. The frozen sections of the adrenals were stained with Scharlach, Sudan III, or the same combined with hæmatoxylin.

CASE I.

Clinical Notes.

M. T. A., aged 47, widow; former occupation a cook. First attack. Admitted to Claybury Asylum on August 10, 1914. Died on December 30, 1914.

According to the certificates, she is rambling about people talking to her and calling her on the telephone. She imagines that she has electricity about her; delusions that people are watching her in her room and from the houses opposite. She answers imaginary voices, and has telephone messages to go to

different places. She imagines also that she has electricity at the back of her head, and thinks her bed is electrified. Her son says she has suffered with delusions for about a week; she imagines people are talking to her from the windows opposite, that telephone wires run through the room to the house opposite, and messages are sent through it. She has an idea that people have put a spell on her, and that she might be poisoned.

State on admission: Fair physique, good nutrition. Lungs normal. Heart's action irregular; pulse 90. Mental condition: She is very noisy and excitable, raving and shouting; rambling and incoherent. She believes she is being electrified. She has visual and aural hallucinations. Hands are broad and spade-like. Face is swollen and pasty, but not œdematous. Her condition suggests a case of myxœdema.

Progress of case, August 17, 1914: She is suffering from confusional insanity; myxœdema. She is *deliberate and slow in response to questions*. Rather restless, very rambling. She has no correct knowledge of time or place, and has hallucinations of sight and hearing. She believes she is strapped down to the electric light, and that she is here to be ill-used and have her circulation and breathing stopped. She has visceral sensory disturbances. She is continually chattering to herself in an incoherent manner. She does not know whether to take her own life or not. Well nourished, but in greatly impaired health.

The patient did not improve, retained her persecutory delusions, and on December 28, 1914, commenced to have seizures. Pupils reacted sluggishly to light and accommodation, and several notes indicate that during the later period of her life she was becoming demented, for she was said to be suffering with general paralysis. She died on December 30, 1914, from bronchitis and emphysema of both lungs.

Autopsy (made by Dr. Mott).

A female, fair physique, body well nourished, little puffiness about the eyelids, no bruises or bed-sores. Linea alba on abdomen. No enlargement of hands or feet. Hair is thin. General appearance is suggestive of hypothyroidism. No external marks of syphilis.

The skull is normal in appearance. Dura mater and pia-arachnoid are not thickened. Stripping of pia-arachnoid without erosion in a normal manner. In the subarachnoid space no excess of fluid. Encephalon is normal in appearance. Convolutions exhibit a somewhat simple pattern. No local softening, not much general wasting. Very slight superficial hæmorrhage on the surface of the pons. *No granulations in the fourth ventricle*. The pituitary body is remarkably larger than the normal; weighed 1.05 gm., reddish. It stands out a little from the sella turcica. The pineal gland is pink and fairly large; weighed 0.25 gm.

Larynx shows marked œdema of the tissue forming the glottis and epiglottis. The thyroid gland is very pale, fibrous, and compact; weighed 33.6 gm. The external parathyroid glands are four in number, of irregular

oblong shape, fairly large, 0·05 gm. to 0·06 gm. Bronchi contain frothy mucous fluid and are congested. Right pleura: No adhesions, no fluid. Left pleura: Slight adhesion at apex, no fluid. Right lung emphysematous, all parts crepitate, congested, œdematous; the left same as the right.

Small bronchi are congested, filled with frothy mucous fluid. Heart muscle is firm, but fairly easily lacerated by the finger-nail. The ventricles are contracted, firm, do not collapse. Atheroma of first degree in arch of aorta more marked in the descending branch.

The liver congested, rather fatty on section. The right kidney: The capsule thickened, somewhat adherent, strips leaving a slightly granular surface. Excessive fat in the pelvis. The left kidney same as the right. The right adrenal is smaller than natural, 5·08 gm., normal on section; the left also normal, 5·25 gm. Abdominal aorta shows a little atheroma. The right ovary is of the size of a cricket ball; section shows this to be due to a dermoid cyst; the left is fibrotic, the surface is corrugated, 3·35 gm. The Wassermann reaction of the serum and of the cerebrospinal fluid was negative.

Cause of Death and other Pathological Conditions.—Œdema glottidis, bronchitis, emphysema of both lungs, hypothyroidism, myxœdema.

Microscopical Examination.

(1) *The Thyroid Gland.*—The vesicular structure of the gland is almost entirely replaced by dense connective tissue, in which marked lymphocyte infiltration is seen, here and there, forming islets of cells. The lymphoid cell masses vary in size and form. In this mass the remnants of the atrophied vesicles are scattered. The epithelial cells of the vesicles have undergone hyaline degeneration. Some few of the remnants of the vesicles contain colloid, which is stained by eosin and picric acid (hæmatoxylin—Van Gieson); other atrophied vesicles contain only disintegrated epithelial cells. Under a high power the fibres of the connective tissue appear swollen and thickened. The epithelial cells have a degenerated hyaline homogeneous appearance. Some slight thickening of the wall of the blood-vessels is also observed (*see* Plate, figs. 1, 2, 3, and fig. 5).

(2) *The Parathyroid Glands.*—In the middle of the section, forming the glandular structure, comparatively abundant principal cells are seen; near the periphery they decrease in number. The converse is the case with the oxyphile cells (Welsh). The capillaries are dilated and sinus-like. Some follicles contain colloidal drops, which are stained with eosin; in the lumen of some follicles can be seen the detritus of the cells.

(3) *The Pituitary Body.*—Some capillaries are remarkably dilated like a sinus, accompanied by thickened connective tissue. The *pars intermedia* is enlarged, and a few large cystic vesicles are seen, and excess of colloid. Throughout the whole section of the glandular portion, especially in the hind-part and near the periphery, are many cyanophile cells which vary in size; in the other part they are either scattered or in groups. There are many

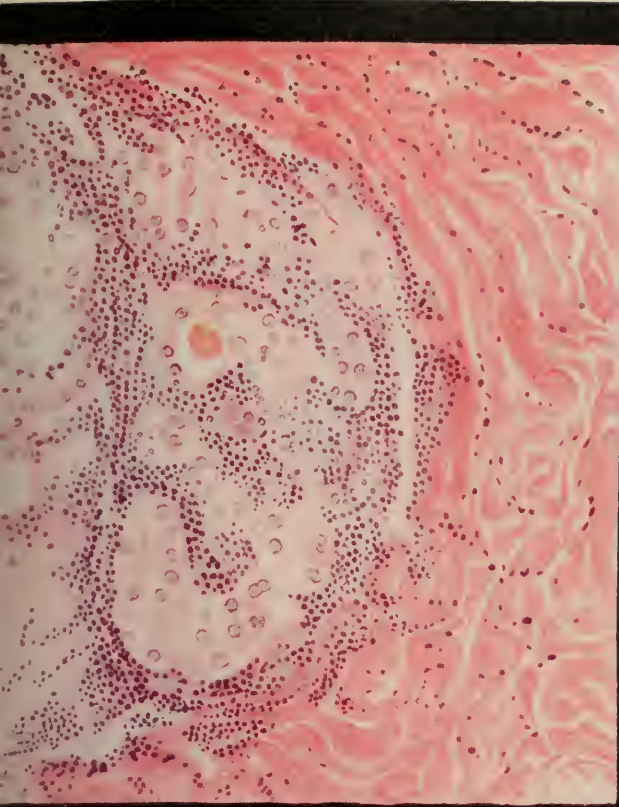


FIG. 1.

Thyroid gland (Case I), Van Gieson hæmatoxylin, showing increase of connective tissue and lymphoid infiltration. The remnants of the gland containing colloid are seen. ($\times 80$.)

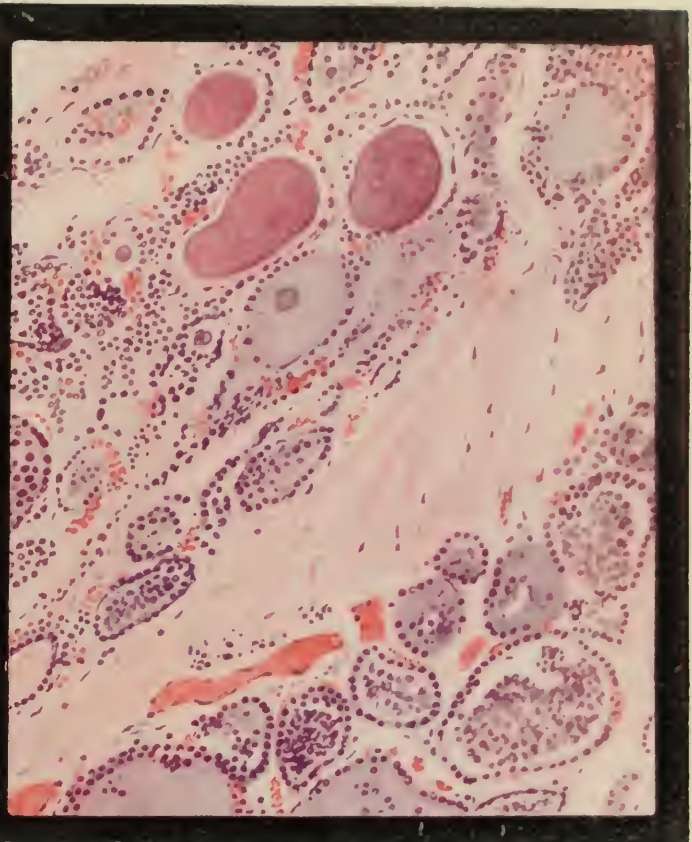


FIG. 4.

Thyroid gland (Case IV), hæmatoxylin-eosin, showing an increase of connective tissue, marked congestion and slight lymphoid infiltration around the vesicles. Débris of epithelium in the vesicles are also seen. Colloid shows a micro-chemical change. ($\times 160$.)

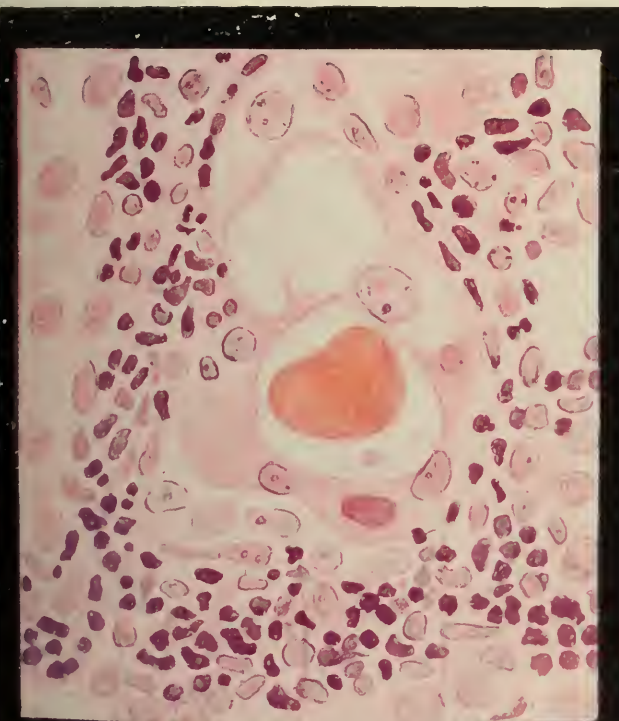


FIG. 2.

Same as fig. 1, showing hyaline degeneration of epithelial cells of the vesicles containing colloid. Lymphocytes and débris of epithelium are also seen. ($\times 330$.)



FIG. 3.

Same as fig. 1, showing degeneration of epithelium of vesicle containing colloid. ($\times 750$.)

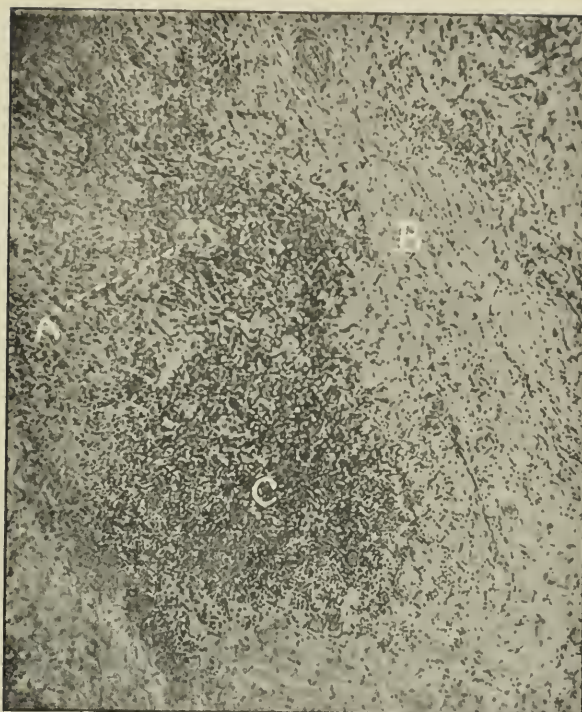


FIG. 5.

Thyroid gland (Case I), hæmatoxylin-eosin. The colloid-containing vesicles have almost disappeared, only a few remnants (A) of the gland structure being left. Replacement of the glandular tissue by fibrous (B) and lymphoid tissue (C) has occurred. ($\times 90$.)



FIG. 6.

Parathyroid gland (Case I), hæmatoxylin-eosin, showing abundant principal cells forming glandular structure. ($\times 200$.)

eosinophile cells, especially in the hind-part of the glandular portion. The principal cells are in fair number; their protoplasm is scanty, clear, or slightly stained with eosin. The lumen of some follicles contain colloid as a drop, stained dark red. A few lymphocytes are scattered in the interstitial tissue of the *pars intermedia*. Some of the cyanophile cells contain a homogeneous clear substance. Generally speaking, both the eosinophile and cyanophile cells are remarkably increased in number in proportion to the principal cells. The posterior portion (*pars nervosa*) appears to show no special changes.

(4) *The Pineal Gland*.—In the middle of the gland is a large fibrotic patch containing a large cyst with a thick fibrotic wall. In the hind-part of the gland there is also a large cyst. In the remaining parts are numerous alveoli, of various sizes, separated from each other by septa of concentric bodies consisting of connective tissue. Near the periphery of the gland earthy salts are seen, which vary in size and shape, stained dark red with hæmatoxylin-eosin. The cysts contain serous fluid with many disintegrated cells.

(5) *The Adrenals*.—The cortex and medullary portions are intact. The lipoid substance is generally diminished in the cells of the cortex. Some of the cells, especially in the zona glomerulosa, contain no lipoid.

(6) *The Ovary*.—The left ovary is fibrous; there are no follicles, and the gland presents the appearance of complete involution. There are seen here and there in the section many small old corpora albicantia and three large old *corpora lutea*, scattered around which are lutein cells.

Résumé.—The thyroid gland shows the most advanced atrophic change with increased connective tissue and considerable accumulation of lymphoid cells. All the epithelial cells have undergone hyaline degeneration, destroying the proper function of the gland. Generally speaking, the thyroid gland shows a pseudo-hypertrophy. The accumulation of the lymphocytes and fibrous tissue suggests a chronic inflammatory change, possibly of toxic origin.

CASE II.

Clinical Notes.

E. M. E., aged 44, married, housewife. Admitted to Claybury Asylum on November 19, 1914, from Tooting Bec Asylum, wherè she had been since November 17, 1914. First attack. Died on December 30, 1914.

According to the certificates she imagines that she is in Kentish Town Hospital, and that she has just arrived. She imagines also that some women are playing tricks on her, and trying to take her husband from her; she has no idea where she was living. She is restless and noisy. She thinks that all her food is poisoned, and she is subject to outbursts of acute excitement, when she is very violent.

State on admission: Fair physique, nutrition poor. Scattered rhonchi in both lungs. Heart and pulse normal. Pupil reflexes are also normal. Knee-jerks are present. Mental condition: Reaction time is increased. She

is unable to answer simple questions. She thinks that the nurse is named "Lizzie" or "Mrs. Knight," and that this is the "Hospital for Cats." Habits dirty. Noisy and abusive at times.

Progress of case (November 26, 1914): She is suffering from confusional insanity, alcoholic (?). She is lost and wandering, making incoherent statements as to a baby being with her in bed and being then stolen. She thinks that she is in the Asylum tavern, Caterham; also that she is in the Council schools, where she has been for some weeks. No idea of time or place; confuses individuals, whom she addresses familiarly. General habits and condition are poor. Feeble and shaky. Looks ill and pallid. Knee-jerks are normal; no pains in legs, but unable to walk without support.

She gradually sank and died from broncho-pneumonia on December 30, 1914.

Autopsy (made by Dr. Mott).

A female, rather emaciated, teeth in upper jaw deficient, palate high and narrow. Linea alba over abdomen and thighs. Over each knee a fibrous growth the size of a walnut.

The skull is normal in appearance. Beneath the scalp is a considerable hæmorrhage which extends beneath the pericranium and appears to be recent. No sign of fracture of calvarium. Pia-arachnoid is opalescent, thickened, pale, stripping fairly readily. The convolutions of the encephalon are rather simple; there is some general wasting, especially of fronto-central regions. No local softening. Striations of the cortex are indistinct. No granulation in the fourth ventricle. Lateral ventricles are dilated. The pituitary body is slightly reddish, rather large, 0.77 gm. The pineal gland is also rather large, 0.12 gm.

The larynx contains a quantity of blood and mucus. The thyroid gland is small, compact, 9.2 gm. There were two external parathyroid glands on both sides. Thorax: Cartilages are not ossified. Pleura: No fluid, no adhesions. The bronchi contains blood-stained mucus. Both lungs show emphysema and broncho-pneumonia. Heart, *nil*, abnormal. Aorta shows early arterio-sclerosis.

The liver very congested, marked by corset. The spleen is enlarged and congested. The right kidney is small, excess of fat is present in the pelvis; the capsule strips leaving a slightly granular surface with cysts. The left kidney shows nothing special. The right adrenal is firm, good colour, 4.25 gm.; the left the same, 4.55 gm. The abdominal aorta shows a few slight nodular enlargements. Throughout the large intestine is seen a thickening of the mucous membrane, but no ulcerations.

The left ovary is bound to uterus by firm adhesions, weight 1.6 gm.; the right also, but less markedly so, weight 1.2 gm. The Wassermann reaction of the cerebrospinal fluid was negative.

Cause of Death and other Pathological Conditions.—Broncho-pneumonia, hypothyroidism, old parametritis, old salpingitis of both sides, grave anæmia.

Microscopical Examination.

(1) *The Thyroid Gland.*—The sections of the several parts show that the gland is divided into many different-sized lobules by dense connective tissue septa. Each lobule is also subdivided into small portions by thickened connective tissue. The gland structure shows, in general, atrophy, and the vesicles vary in size considerably. The vesicles are remarkably diminished in size. Some vesicles show a papillomatous change, and the epithelial cells are, in general, cubical; the nuclei are nearly round, containing uniform fine granules. But in some vesicles the epithelial lining is remarkably flattened,



FIG. 7.

Thyroid gland (Case II), hæmatoxylin-eosin, showing an increase of fibrous tissue (A) and lymphoid infiltration (B), with a corresponding atrophy of the vesicles, which vary in size. The epithelial cells are flattened; some of them under a higher magnification show commencing degeneration. ($\times 90$.)

and the nuclei are long and narrow, and surrounded by scanty cytoplasm. A few epithelial cells show fine eosinophile granules in the cytoplasm. Around the vesicles there are accumulations of lymphocytes. Some portions of the fluid consist almost entirely of lymphocytes, in which small vesicles are here and there scattered. Some vesicles contain colloid, stained feebly or deeply with eosin. The smaller vesicles are often filled with desquamating epithelial cells. Some slight thickening of blood-vessels is seen (*see fig. 7*).

(2) *The Parathyroid Glands*.—Abundant principal cells are seen in the middle and margin of the gland. Many oxyphile cells are also seen as strands, or scattered throughout the section. Many of the follicles contain colloid, stained deeply with eosin.

(3) *The Pituitary Body*.—In the *pars intermedia* many large vesicles are seen: it contains colloid, stained with eosin. The *pars nervosa* appears to show no special changes. Certain follicles of the glandular portion are dilated and filled with colloid, stained deeply with eosin. The eosinophile cells are abundant in number in the middle and in the neighbourhood of the *pars intermedia*. In general, the cyanophile cells are increased and have a contrary arrangement to the eosinophile cells. Nearly all the follicles in the anterior

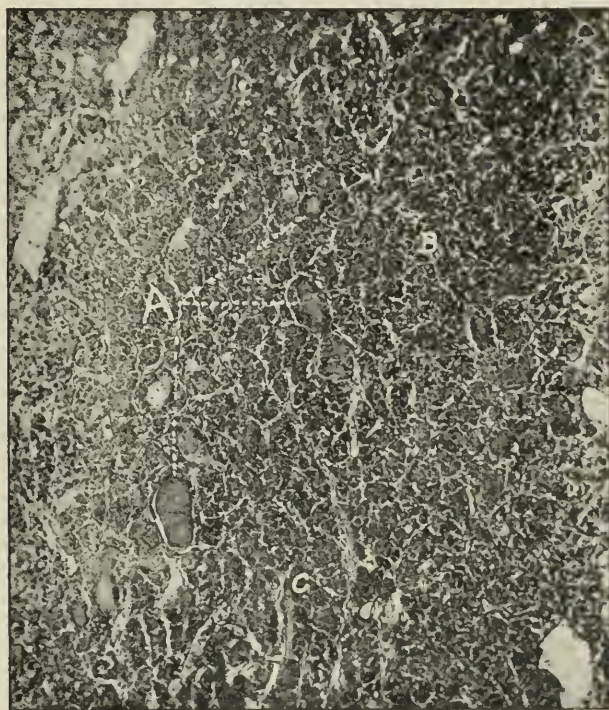


FIG. 8.

Pituitary body (Case II), hæmatoxylin-eosin, showing an enlargement of the follicles (A) containing colloid. Abundant eosinophile (B) and cyanophile (C) cells are also seen. ($\times 90$.)

part of the glandular portion are composed of cyanophile cells only; but in other parts of the gland the two kinds of cells and the principal cells are seen in the follicles. The principal cells are fairly numerous in number (*see* fig. 8).

(4) *The Pineal Gland*.—The section shows a complete involution of the gland and contains many masses of brain-sand, which vary in size and shape, as in Case I. There is also an increase of connective tissue, even more than in Case I.

(5) *The Adrenals*.—The cortex is stained unevenly with Scharlach. Some groups of the cortex cells are stained feebly; on the other hand, there are

deeply stained groups of cells. In general, the lipoid substance is considerably diminished.

(6) *The Ovaries*.—Both ovaries are, generally speaking, fibrotic. There are a few corpora albicantia and two small corpora lutea. Neither lutein cells nor follicles are seen. The appearances suggest an early complete involution.

Résumé.—The thyroid gland shows atrophy of the glandular substance associated with the appearances of a chronic inflammatory change in the interstitial tissue, as shown by the lymphocytic proliferation and fibrosis. As in Case I, the pituitary was enlarged, and an excess of colloid shown.

CASE III.

Clinical Notes.

A. J., aged 67, married, housewife, former occupation a dancer. Admitted to Claybury Asylum on June 8, 1906. The age on first attack was 58. Died on January 15, 1915.

According to the certificates she says a baby has been killed and that she is going to be hanged for it. She also fancies that other inmates are continually interfering with her, and that the nurse has been putting poison in her tea. She imagines that the people are calling out to her and answers them. She is troublesome and keeps undressing herself, fancying there are rats about her.

State on admission: Venules on face. Tremor of tongue. Heart is not enlarged; systolic sound at apex conducted outwards. Pulse is regular. Pupils are equal, resist sluggishly to light. Knee-jerks are present. Mental condition: She speaks slowly and indistinctly. Marked tremor of face, hands and tongue. Her memory is poor; no idea of time and place. Lost and confused; very emotional.

Progress of case (June 14, 1906): She is suffering from melancholia, alcoholic (?) Her memory is greatly impaired. She thinks that she is in a music-hall, and that to-day is March 10. Speech tremulous; difficult to understand. Tremor of facial muscles during speech. Wanders aimlessly about. Very depressed, mind weakened. Poor health and condition. Right pupil is slightly larger than the left, and reaction to light uncertain. Knee-jerks are present.

May 5, 1907: She is suffering from dementia (alcoholic? paralytica?). Slight general impairment of the higher mental faculties, especially those of understanding and reason, in particular in regard to her present relationships to her environment, and a weakening of the memory. Conduct good; works well under supervision. No special propensity to forgetfulness. Sanguine, even optimistic about herself. Fair health and condition. Fairly well marked tremors of lips and tongue. Pupils equal, they react to light, but sluggishly to accommodation. Knee-jerks are somewhat exaggerated.

May 5, 1908: She is suffering from general paralysis of the insane. She

is facile and emotional. Her memory is impaired, and she asks the same questions day after day. At times cries for no obvious reason. Fair health and bodily condition.

She continued to be tremulous, and gradually became demented and more emotional and incoherent. On January 13, 1915, she appeared dazed and lost, and was put to bed. On January 14 she had a number of seizures, and died from acute bronchitis and emphysema on January 15, 1915.

Autopsy (made by Dr. Mott).

A female, well nourished, good physique. The hands are rather large and œdematous, also the feet. No œdema elsewhere. The face is rather puffy and œdematous.

The skull is thicker and denser than natural. The dura mater is adherent. The subdural space contains an excess of fluid, the subarachnoid space also an excess of fluid. Encephalon: The convolutions are rather simple in pattern. A little general wasting. No local softening. No erosions on stripping the pia-arachnoid. The white matter is œdematous. No signs of granulation in the fourth ventricle. The pituitary body is considerably enlarged, 1.2 gm., especially its anterior part. The *sella turcica* is very large and the posterior clinoid processes have been absorbed. The pineal gland is comparatively small, 0.07 gm.

The larynx and the trachea are congested. The thyroid gland is small, pale in colour, 12.6 gm. There are four parathyroid glands, two on each side. Both pleura are free from adhesions and fluid. Bronchi are very congested, extending to the smallest tube. Both lungs are congested and œdematous. The left lung is emphysematous. The heart shows hypertrophy of the left ventricle, of fair colour; no valvular disease. The aorta shows nothing special.

The liver is small, congested, fatty. The spleen is large and pulpy. Both kidneys *nil*. Both adrenals are disintegrated in the centre—the right weighed 4.95 gm., the left 4.65 gm. The right ovary is small, shrunken and hard, 1.4 gm.; the left is destroyed by very old adhesive salpingitis. The Wassermann reaction of the serum and of the cerebrospinal fluid was negative.

Cause of Death and other Pathological Conditions.—Acute bronchitis, emphysema, chronic Bright's disease, hypothyroidism, old adhesive salpingitis.

Microscopical Examination.

(1) *The Thyroid Gland.*—In general the sections show a marked hyperæmia and an increase of the intervesicular connective tissue. The vesicles contain colloid stained purple with hæmatoxylin-eosin, but most of them contain only débris of epithelium, stained dark blue by hæmatoxylin. The capillaries around the vesicles show marked congestion. Hæmorrhages are seen in certain parts. The epithelial cells are generally flattened; the nuclei are round, containing fine granules. A few epithelial cells contain fine eosinophile granules in the cytoplasm. There is slight lymphoid cell infiltration of the

interstitial tissue. There is a desquamation of the cell lining of the vesicles. In some of the vesicles, embedded in the ordinary colloid, there are several large circular bodies which are deeply stained with hæmatoxylin. They have no structure and appear to be colloidal in nature. The blood-vessels of the gland show slight thickening of the tunica intima (*see* fig. 9).

(2) *The Parathyroid Glands.*—The whole transection of the gland shows a number of hollow spaces which appear to be vesicles, similar in appearance to the fat vesicles of the exterior of the gland. Many sections also show patches of various degrees of coagulation necrosis of the epithelium. These necrotic cells exist in patches of variable size throughout the gland. In the

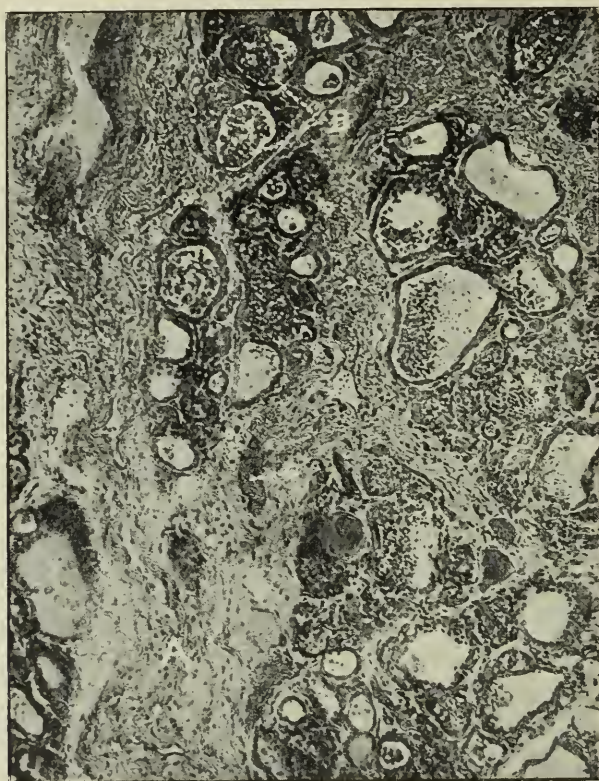


FIG. 9.

Thyroid gland (Case III), hæmatoxylin-eosin, showing a marked hyperæmia and an increase of the intervesicular connective tissue (A). Some vesicles contain colloid, or colloid mingled with débris of cells (B), and others are filled with débris of cells only (C). The colloid shows microchemical change, stained with eosin or hæmatoxylin. A blood-vessel is seen here and there. ($\times 90$.)

lumen of the follicle colloid is seen, here and there, in the section; in one part there is a comparatively large cyst, filled with colloid, which is stained deeply with eosin. The whole section shows a marked increase of the oxyphile cells and a relatively less number of the principal cells. The right upper parathyroid gland contains more colloid than the other (*see* fig. 10).

(3) *The Pituitary Body.*—In general the glandular portion is enlarged, and



FIG. 10.

Parathyroid gland (Case III), hæmatoxylin-eosin. In the middle the patch of coagulation necrosis (A) is seen. Around the patch there are many oxyphil cells. ($\times 90$.)

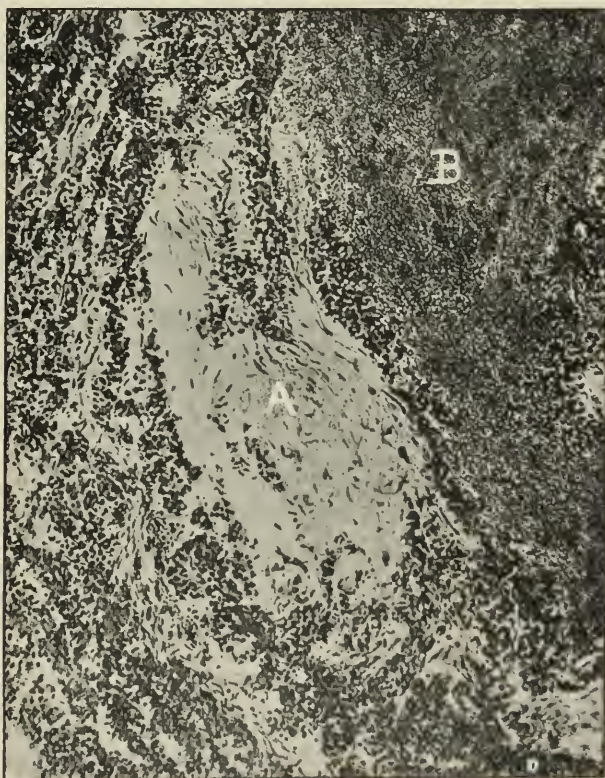


FIG. 11.

Pituitary body (Case III), hæmatoxylin-eosin, showing in the middle a patch of connective tissue (A), and on the right a hæmorrhagic patch (B) is seen. ($\times 90$.)

the middle of it shows a fairly large patch of connective tissue. In the other parts the connective tissue is also increased. Close to the patch of connective tissue there is also a large hæmorrhagic patch, apparently of recent origin, which can be seen even by the naked eye. Around the hæmorrhagic patch there is a slight lymphoid cell infiltration. The other parts show a slight hyperæmia. Many follicles in the *pars anterior* are larger than usual and contain colloid. There are also abundant eosinophile cells, especially in the posterior part. The cyanophile cells are increased in number, and are seen most in the front part. The intermediate and the posterior portions are also very congested (*see fig. 11*). The *pars intermedia* did not show, as in Cases I and II, a marked increase of colloid.

(4) *The Pineal Gland*.—The whole section shows a marked thickening of the inter-lobular connective tissue. Otherwise there is no special change.

(5) *The Adrenals*.—The cortex sections show unevenly stained patches with Scharlach. Certain cortex cells are loaded with abundant lipoid, but most of them contain scanty lipoid.

The ovary on the right side is fibrous throughout, and shows advanced involution. Corpora albicantia are seen, but no lutein cells were observable.

Résumé.—The thyroid gland shows atrophy, with a much less degree of interstitial change than the two previous cases. The hyperæmia of the gland may be due to the acute bronchitis, and congestion of the lungs and other organs. With regard to the necrotic process of the cells of the parathyroid glands, it is difficult to come to any conclusion.

CASE IV.

Clinical Notes.

G. M. P., aged 51, married, housewife. Admitted to Claybury Asylum on December 10, 1914. First attack. Died on January 2, 1915.

According to the certificates she is confused. Her memory is poor. She imagines that other inmates of the ward are her relations. She calls to imaginary people in the ward to bring her beer. Her husband stated that she has been drinking heavily for some time.

State on admission: Fair physique, nutrition poor. Lungs are normal. Artery wall thickened. Pulse 72, collapsing type. Heart sounds at apex; first sound systolic murmur, second sound loud, late, diastolic murmur. The liver is hard, 2 in. below the costal margin. Urine shows a trace of albumin. Pupil reflexes are present. Knee-jerks are also present. Varicose veins of both legs. Mental condition: Mode of speech is slow. Reaction time is increased. She is unable to give an account of herself. Her memory is lost. She makes mistakes in identity. No delusions or hallucinations. Admits alcoholic excess.

Progress of case (December 17, 1914): She is suffering from confusional insanity. She is depressed, confused, and lost. She has no knowledge of

time and place. Slow to answer questions, dull, and complains of a giddy feeling. She makes imaginary journeys and thinks she came up to see me at the Southend Hospital yesterday, which is not the case. She is helpless and has defective habits. Poor general health and condition.

The patient gradually became worse and died on January 2, 1915, from broncho-pneumonia.

Autopsy (made by Dr. Mott).

Nutrition good. A flush on the cheeks, no venules on the nose. No bruises or bed-sores. The hands are not enlarged. Varicose veins. "The facial expression is somewhat suggestive of cachexia thyreopriva." No external marks of syphilis.

The dura mater is not adherent, no deposit. The subdural space contains no excess of fluid. The pia-arachnoid shows no marked thickening, not stripping readily. The subarachnoid space contains also no excess of fluid. The encephalon: The pattern is very simple, both frontal and parietal lobes being deficient suggests a high-grade imbecile. No local softenings or atrophy. The striation of the line of Gennari is quite distinct. The pituitary body is of normal colour, weighing 0.48 gm. The pineal gland is small, 0.06 gm.

The trachea is congested; the larynx shows post-mortem staining congestion. The thyroid gland is reddish, small, 7.49 gm. The parathyroid glands are two in number at both sides. All parts of the right lung crepitate; the lower lobe is congested and œdematous, the upper lobe is emphysematous and congested. The lower lobe of the left lung is solid, and shows well-marked patches of broncho-pneumonia; the upper lobe is emphysematous and congested. The heart muscle substance is of fairly good colour. No atheroma of aorta.

The liver is marked by tight lacing, firm, and suggests an early fibrosis, marked by fatty degeneration. The spleen is rather pulpy. Both kidneys are somewhat congested, the capsule is slightly adherent. Both adrenals are disintegrated, the right is 3.98 gm. and the left 4.73 gm. No evidence of salpingitis. Both ovaries are flattened, shrunken, and corrugated; the right weighed 1.75 gm., the left 1.55 gm. The Wassermann reaction of the serum and of the cerebrospinal fluid was negative.

Cause of Death and other Pathological Conditions.—Broncho-pneumonia, hypothyroidism.

Microscopic Examination.

(1) *The Thyroid Gland.*—The whole section shows an increase of connective tissue, marked congestion, but no lymphoid infiltration. The vesicles vary in shape and size, and contain colloid, which stains slightly or dark blue with hæmatoxylin, or purple with hæmatoxylin-eosin. The colloid is mingled, in many vesicles, with the detritus of the epithelial cells. The epithelial cells are cuboid in form; the nuclei are round, containing fine granules. The

protoplasm of many cells contains fine eosinophile granules. In many of the colloid masses round spaces like a vacuole are present. Some vesicles are entirely filled with hyaline débris. A desquamation of the lining of the epithelial cell is seen in many vesicles (*see* Plate, fig. 4, and fig. 12).

(2) *The Parathyroid Glands.*—The right gland shows, in general, abundant principal cells, which are decreased in number at the periphery. The left shows, in general, a decrease of the principal cells and an increase of the oxyphile cells. The group of cells near the periphery of the left gland is composed chiefly of oxyphile cells. In a few follicles of the glands colloid is seen, stained bright red with eosin. In the left parathyroid gland fatty infiltration

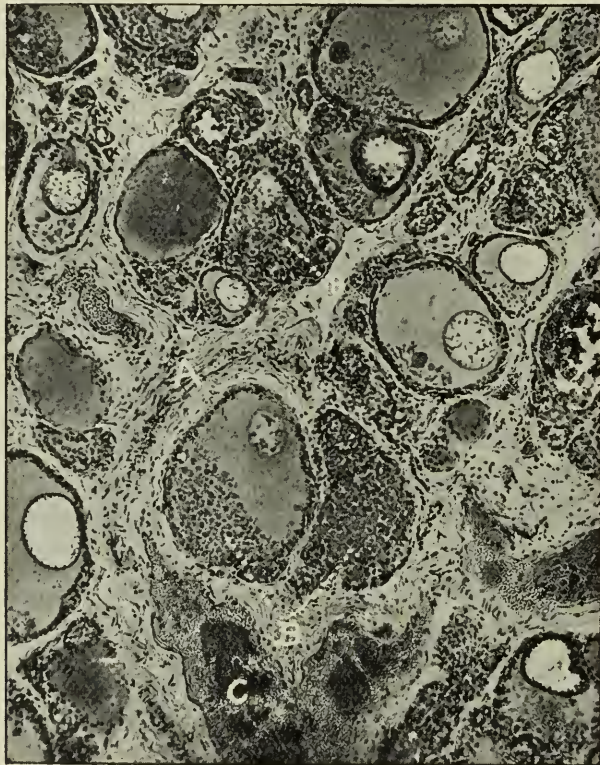


FIG. 12.

Thyroid gland (Case IV), hæmatoxylin-eosin, showing an increase of connective tissue (A), marked congestion, and slight lymphoid infiltration around the vesicles. Epithelial cells are flattened. The vesicles contain colloid, which shows microchemical change, mingled with débris of cells (B). A blood-vessel (C) is seen here and there. ($\times 90$.)

is seen, which extends inwards to the glandular mass, and like islands, separates the glandular structures in many parts. Near the periphery, and in some deeper portions of the gland, patches of the watery-clear cells (Getzowa) are here and there seen (*see* fig. 13).

(3) *The Pituitary Body.*—The infundibulum, the anterior, the posterior and the intermediate portions are very congested. Nearly all the capillaries

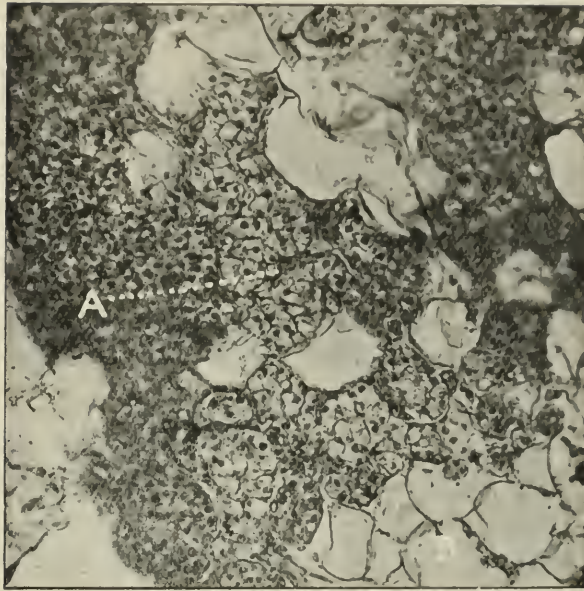


FIG. 13.

Parathyroid gland (Case IV), hæmatoxylin-eosin, showing "watery clear" cells (A). There is a large amount of interstitial adipose tissue (B). ($\times 200$.)

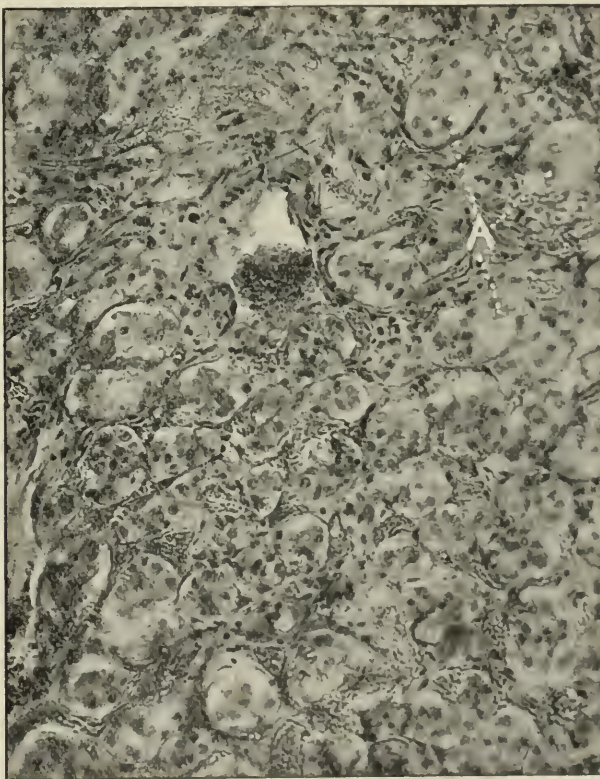


FIG. 14.

Pituitary body (Case IV), hæmatoxylin-eosin, showing the many degenerated cells in the hind-part of the pars anterior (A). There is marked hyperæmia. ($\times 200$.)

are somewhat dilated. The *pars intermedia* is thickened, and large colloid-containing vesicles are seen. In one part of the glandular portion there is a small hæmorrhagic patch. The glandular *pars anterior* contains abundant eosinophile cells, especially near the *pars intermedia*. There are many degenerated cells in the hind-part of the glandular portion. The cyanophile cells are also seen in great number. In some parts the cells are in a state of commencing degeneration. In the glandular portion many follicles contain colloid (*see fig. 14*).

(4) *The Pineal Gland*.—In the hind-part of the gland there are many masses of brain-sand of various size, stained with eosin. The intralobular connective tissue is somewhat thickened.

(5) *The Adrenals*.—The cells of the cortex of both right and left adrenals which take the Scharlach lipid stain are relatively few in number.

(6) *The Ovaries*.—Both right and left ovaries are fibrotic. There are many corpora albicantia and a somewhat large corpus luteum. No lutein cells are seen.

Résumé.—The changes in the thyroid gland differ from those previously described from the fact that although there is an atrophy of the glandular structure there is no evidence of a chronic toxic inflammatory process in the interstitial tissue, as no lymphocytic infiltration is observable.

CONCLUSION.

In the first two cases the changes of the thyroid gland are more advanced than the others. All the changes which occurred in the thyroid glands in the first three cases may have had a toxic origin. These changes of the thyroid gland, in which there were marked mental confusion and depression, correspond entirely with the changes observed by Dr. Mott and Dr. Brun in the third case described by them.¹

The enlargement of the pituitary body, especially of its glandular portion in conjunction with the changes of the thyroid gland, has been observed by Rozowitch, Schonemann, Ponfick, Boyce and Beadles, Mott and Brun. In the first three cases the pituitary body, especially its glandular portion, is much enlarged. In Case IV the pituitary body is, on the contrary, comparatively small. From this it may probably be inferred that it may become larger, according to the progress of the changes in the thyroid gland.

In the first two cases the microscopic examination of the pituitary body showed a marked activity of function. In Case III the pituitary body was very large, showing, in general, an activity of function, though the *pars anterior* shows commencing atrophy, marked by an increase

¹ *Proceedings*, 1913, vi, p. 89.

of connective tissue. In Case IV it shows in one part a state of commencing degeneration of the cells, but other parts show a marked activity of function.

Although the parathyroid gland is closely attached to the thyroid gland, it did not show an increase of connective tissue or lymphoid cell infiltration met with in the thyroid gland in the first three cases. The fact is of some importance in showing that a toxic condition of the blood only is not responsible for the changes noted in the thyroid gland, and supporting the view that this condition is not a general one, but due to progressive changes in the gland of the nature of a thyroid insufficiency. This thyroid insufficiency was most marked in the first case. In Cases I and II the parathyroid gland suggests an increase of function; on the contrary, in Case III it shows, here and there, a necrotic process of the cells, suggesting that the function, in general, is lessened. In Case IV the right parathyroid gland shows an increased functional activity, while the left shows a lessened function. It is difficult to form any conclusion about the correlation between the thyroid and parathyroid glands from these histological observations.

The pineal glands of all cases show noteworthy changes which could not be definitely correlated with the changes of other glands or mental disorder.

The four cases showed under Scharlach staining a deficiency of lipoid in the cortex of the adrenals; but on reference to the notes of post-mortem examinations it was found that the patients had died of acute infective disease—viz., broncho-pneumonia—which is the most frequent immediate cause of death of the insane. Elliott has shown that the lipoid is diminished in cases of death from infective disease.

In the first three cases of hypothyroidism, the patients during life had suffered with ovarian disease. It may be remarked that these were the cases in which there was not merely a glandular atrophy, but there was also a marked chronic inflammatory interstitial change, and in these three cases Dr. Mott has found the perinuclear chromatolysis of the ganglion cells of the central nervous system, similar to those previously described by him in conjunction with Dr. Brun.

The systematic examination of all the ductless glands in these cases excludes the probability that changes in any other ductless glands than the thyroid can be held accountable for the mental symptoms and the histological changes found in the central nervous system.

It is of interest to note that examination of the blood and cerebro-spinal fluid in these cases of hypothyroidism did not yield a positive

Wassermann reaction, and therefore syphilis as a cause can probably be excluded.

Since this communication was read I have examined the ductless glands and reproductive organs of a male, No. 43, Table I, in which the thyroid gland weighed 11.5 gm. The thyroid showed a considerable diminution of colloidal vesicles and a fibrotic atrophy, but there was no lymphocytic infiltration as seen in Cases I, II, and III. This case showed no increase in size of the pituitary body. Both testicles exhibited fibrotic atrophy and complete destruction of the glandular structure. The spermatic tubules were entirely replaced by fibrous tissue. The case is of interest in the fact that Dr. Mott finds no evidence of the characteristic universal perinuclear chromatolysis (*vide p. 59*).

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**Microscopic Examination of the Central Nervous System in
Three Cases of Spontaneous Hypothyroidism in relation
to a Type of Insanity.**

By F. W. MOTT, M.D., F.R.S., Hon. LL.D.Edin.

INTRODUCTION.

INCLUDED among the sixty female cases recorded in Dr. Kojima's detailed account of the condition of the ductless glands are three in which I have investigated the condition of the central nervous system, with a view to seeing if the microscopic changes are essentially similar to changes which I have previously described in conjunction with Dr. Brun, and published in the *Proceedings of the Royal Society of Medicine*, Section of Psychiatry, 1913, vi, pp. 75-100.

The three cases already recorded occurred in women of middle age, and the three I am about to refer to occurred also in women about the climacterium. The complete clinical notes of the cases are given by Dr. Kojima on pp. 38-50. An attempt will be made to correlate the psychic syndrome with the cortical changes observed in these cases.

MICROSCOPIC CHANGES IN THE CENTRAL NERVOUS SYSTEM.

The brains were hardened in formalin; sections of 5μ were cut from blocks embedded in paraffin and stained by the Nissl method; other sections were stained by Ranke's Victorian blue method. Briefly stated, the changes observed were similar in character to those already described by Dr. Brun and myself.

There is a universal chromatolytic change in the cells of the central nervous system, sparing no system or group of neurones entirely. The changes were very marked in the bulb, especially affected were the smaller cells of the autonomic nuclei—e.g., the vagus and glosso-pharyngeal. In the cerebellum the Purkinje cells show less chromatolysis than the large motor cells of the medulla oblongata, spinal cord and Betz cells, and there is no increase of the glia tissue (*vide* figs. 2, 5, and 6). These three asylum cases exhibited much more marked changes in the cells of the cortex than the two hospital cases previously described, and more than the asylum case McC., previously recorded.

This latter case showed less pronounced mental symptoms than the three cases recently investigated, consequently it is not surprising to find that in the cortex of the cases now to be considered there were found more extensive cell changes of the cortex. An attempt will now be made to show a correlation of the mental symptoms with the changes found in the cortex. Unfortunately, the notes do not permit correlation of the changes in the bulb with the symptoms indicative of affection of the autonomic nuclei as in the hospital cases. Still, the main object of this communication is to show that there is a type of insanity occurring in women about the climacterium, in which a manic-depressive condition, associated with mental confusion, hallucinations, delusions mainly of persecution, loss of memory of recent events, and terminating in dementia, may arise as a result of a particular form of hypothyroidism. This hypothyroidism is characterised (1) by an atrophy of the glandular structure of the thyroid, interstitial fibrous hyperplasia and abundant infiltration of the same with lymphocytes; a condition of chronic inflammation arising from a toxic condition, probably local in its source, as the adjacent parathyroids show no such change. (2) By an increase in weight of the pituitary gland, and usually abundant colloid in the *pars intermedia*, which may be regarded as evidence of thyroid insufficiency. All four asylum cases of hypothyroidism in which the pituitary body was examined showed increased size of the pituitary and excess of colloid.

As a control it may be stated that the characteristic perinuclear chromatolysis of the nerve cells found in these four cases was not discovered in two cases of simple atrophy of the thyroid gland—viz., Case IV, and the male alluded to on p. 56. Examination of the thyroid gland in these two cases showed still normal colloidal vesicles, and vesicles filled with epithelium or degenerated epithelium; and although there was an increase of the interstitial fibrous tissue, there was no lymphocytic infiltration indicative of a local toxic inflammation; neither was there any increase of weight of the pituitary, nor excess of colloid observed in section of this gland.

It is permissible, therefore, to correlate the changes in the nervous system with this particular form of hypothyroidism, in which the gland, as in Case I, has undergone a pseudo-hypertrophy, or Cases II and III, in which the gland is considerably below the average weight. The increase of colloid in the pituitary may be regarded as an index of the extreme degree of hypothyroidism, but further observations are required to settle this point. If the conclusion of Hering and Cushing be

accepted, that the colloid of the pituitary (*pars intermedia*) passes into the cerebrospinal fluid, then this excess may produce a functional disturbance of the nerve cells, or, as seems more probable, the thyroid insufficiency may be directly correlated with the deficiency of the basophile substance of the nerve cells. In the case of McK., previously described, Brun and I showed that the small posterior spinal ganglion cells were especially affected as well as those of the central nervous system. In any future case that I may have the opportunity of examining I shall examine the sympathetic ganglia.



FIG. 1.

Section of motor cortex pyramidal layer. (× 200.)

EXAMINATION OF THE CORTEX CEREBRI OF CASE I, STAINED
BY NISSL METHOD.

All the cells of the pyramidal layer under a low power show a deficient basophile staining; the cells are stained a pale blue. Obviously the stain is paler in all the cells around the nucleus—perinuclear chromatolysis. Many seem to have lost their apical processes, and no longer retain their pyramidal shape (*vide* fig. 1). There is still evidence of the existence of the columns of Meynert. Examination with an oil-immersion lens does not show a single normal cell. There

is an obvious deficiency of basophile staining, especially around the nucleus; the edges of the cells are ragged; in many the processes are broken off. There are no Nissl blocks, only a basophile staining dust seen generally at the periphery of the cell. The nucleolus is stained

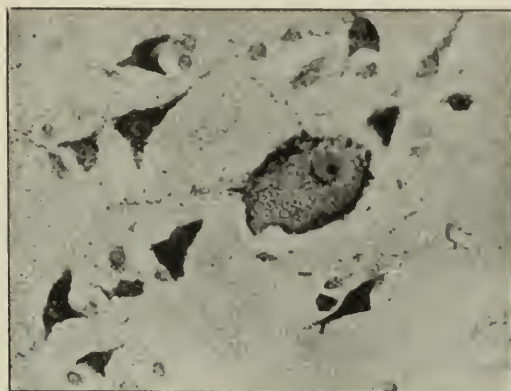


FIG. 2.

Betz cell of motor cortex. ($\times 300$.)

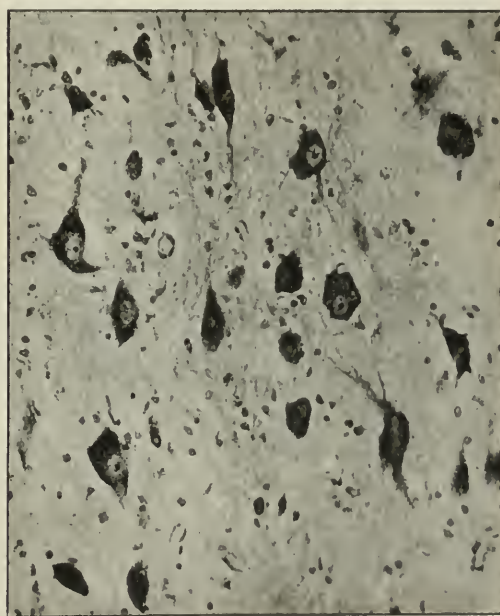


FIG. 3.

Eighth nucleus: A group of cells less affected than is usually the case, but some have an eccentric nucleus and some perinuclear chromatolysis. ($\times 250$.)

deeply; the nuclear membrane and nuclear substance are almost invisible, or only faintly visible and capable of differentiation from the rest of the cells in a large proportion of those examined. In a large number of the

cells there are scattered through the cytoplasm very fine pale yellow round granules, surrounded by uniform pale blue-stained substance. These are lipochrome granules; they are stained by Scharlach, and the great majority of the cells contain some in great or less quantity. These pigmented granules are found in the cells of all old people and are not of much significance in the special pathological condition under consideration.

The changes above noted are just as marked in the deeper layer of

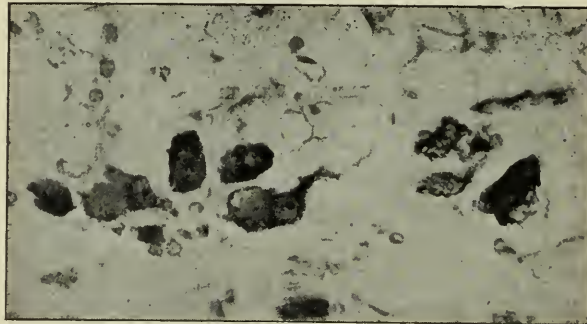


FIG. 4.

A few cells of the eleventh nucleus close to the median raphe. ($\times 250$.)



FIG. 5.

Betz cell of motor cortex (Case II) showing eccentric nucleus. Absence of Nissl granules and processes broken off. Three pyramidal cells are also seen; two exhibit marked perinuclear chromatolysis, one is surrounded by satellite cells. ($\times 450$.)

polymorphic cells of the cortex as in the pyramidal layers. Some of the largest pyramids and some of the Betz cells show still some basophile substance, taking the form of an imperfect Nissl granulation pattern at the periphery of the cell and on the processes (*vide* fig. 2).

Medulla Oblongata.—Every cell in the section, including those of

the olive, shows some degree of chromatolysis; the great majority of cells show an advanced perinuclear chromatolysis, the Nissl granules

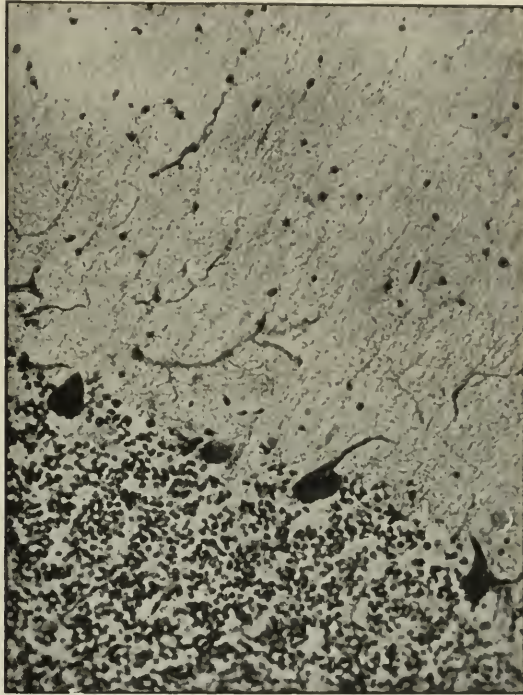


FIG. 6.

Section of the cerebellum stained by Ranke's Victorian blue method for neuroglia. The Purkinje cells are deeply stained, and there is no excess of glia tissue. Compare this with fig. 10. ($\times 250$.)



FIG. 7.

Section of superficial layer of motor cortex stained by Ranke's method. Some excess of glia tissue is seen. ($\times 250$.)

being replaced by a fine basophile staining dust. A few of the large cells of the somatic motor nuclei have a Nissl pattern, but even these

show perinuclear disappearance of the granules. The cells of the various nuclei are not equally affected. Thus the eighth nucleus shows cells exhibiting less perinuclear chromatolysis than others (*vide* fig. 3). The smaller cells show, as a rule, the greater chromatolytic change. They are often with ragged edges, or the processes are broken off (*vide* fig. 4). Still, as in the cases previously described, the bio-chemical change is a general one affecting the whole of the neurones of the central nervous system.

The other two cases (II and III) show the same general perinuclear chromatolysis. Although there does not appear to be an actual deficiency in number of the cortical cells, yet microscopic examination

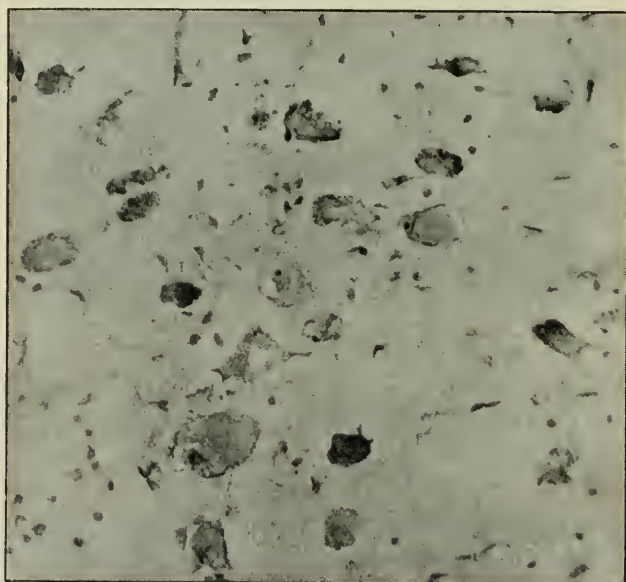


FIG. 8.

Roller's nucleus : Medulla oblongata showing advanced chromatolysis of the cells. ($\times 250$.)

with an oil-immersion lens shows that none of the cells can be regarded as of normal appearance, either in the medulla oblongata or the cortex, but they are by no means all equally affected. As a rule the small cells of the cortex show more change, but this may be due to the fact that they normally contain less basophile substance. Still, examination of the large Betz cells (*vide* fig. 5) shows a striking contrast to the cells of Purkinje (*vide* fig. 6). Many cells of the cortex and of the medulla show dendrons or dendrites broken off. The nucleus is eccentric and the basophile substance has almost disappeared in many of the cells. The satellite cells have greatly increased around these decaying

neurones (*vide* fig. 5). The cortex, stained by Ranke's Victorian blue method, shows that where the nerve cells of the cortex are most affected there is an increase of neuroglia tissue; this glia hyperplasia is not equally distributed, and is most obvious in the tangential layer of the cortex (*vide* fig. 7). In Case II there is apparently hyperchromatosis of the cells of the olive, whereas other groups of small cells, such as those of the vagus and glosso-pharyngeal nuclei, show a very marked chromatolysis. Sections stained by the Heidenhain hæmatoxylin method show a large number of cells stained pink instead of blue. These are the cells which by the Nissl method show a marked chromatolysis.



FIG. 9.

Cells of nucleus ambiguus showing perinuclear chromatolysis. ($\times 250$.)

In Case II groups of neurones in the medulla oblongata show very marked chromatolytic changes, and many cells appear to have their processes broken off (*vide* figs. 8 and 9). Sections stained with Victorian blue show an apparent increase of the glia tissue around these cells (*vide* fig. 10). The medulla and cortex of this were also stained by the Bielchowsky silver method. It appeared that the superior layer of small pyramids and the large Betz cells presented the greatest degree of fibrillary change. Many of the cells of the superior pyramidal layer had entirely lost their pyramidal shape. Still, a very marked contrast exists between the appearances presented by the cortical layer of cells in hypothyroidism and general paralysis or organic dementia (*vide*

fig. 11). This leads one to think that the symptoms are largely due to a lack of functional energy which may be correlated with the marked disappearance of the Nissl substance (kinetoplasm), possibly associated with a toxic condition.

SIGNIFICANCE OF THE NISSL GRANULES AND CHROMATOLYSIS
IN HYPOTHYROIDISM.

The perinuclear chromatolysis found in hypothyroidism is not specific, it is met with in other toxic conditions—e.g., lead encephal-

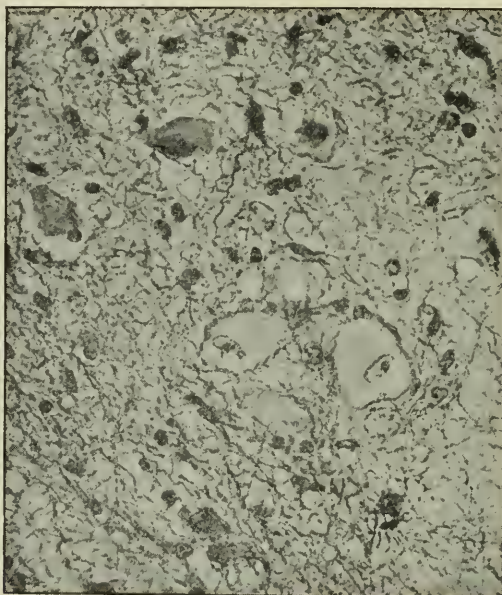


FIG. 10.

Group of cells of medulla oblongata stained by Ranke's Victorian blue method. Most of the ganglion cells are unstained except the nucleolus; the glia cells and glia fibril network show hyperplasia. Glia proliferation, however, is exceptional. ($\times 300$.)

itis and alcoholic psychosis, and similar changes may be seen in experimental anæmia and following sections of the axis cylinder of a nerve cell. The Nissl granules of basophile substance, as I pointed out in 1900 (the Croonian Lectures, "On the Degeneration of the Neurone"), probably represent kinetoplasm, but in the living cell this kinetoplasm does not take the shape of granules forming a pattern in the cell. Nevertheless, the amount of this basophile staining substance in the form of Nissl granules may be regarded as evidence of the amount of energy substance (neuro-potential) which the cells possessed during life.

In the healthy cell it is continually undergoing disintegration and automatic re-integration. When the cell is damaged by injury of its processes, as, for example, section of the axon, experimental anæmia, toxic conditions of the blood, or hypothyroidism, metabolic equilibrium is no longer maintained; its osmotic surface tension is altered and water passes into the cell, causing it to swell, displacing the nucleus and causing a chromatolytic appearance. The disappearance or partial disappearance of the kinetoplasm in the autonomic nuclei of the bulb, Dr. Brun and I correlated with cardiac and other bulbar symptoms

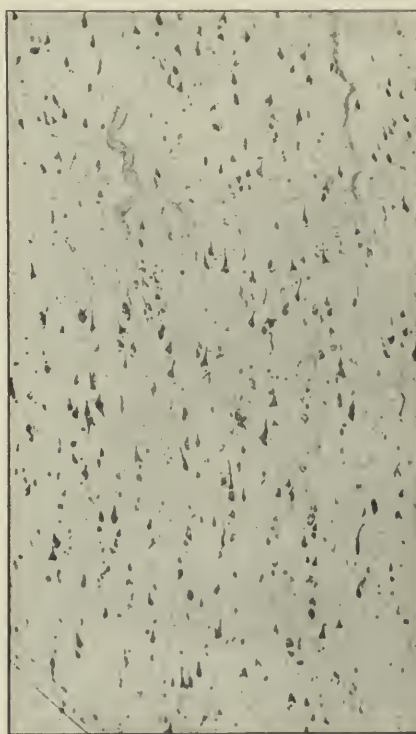


FIG. 11.

Section of cortical pyramidal layer (Case III). There are no signs of increased vascularity or diminution in the number of ganglion cells, nor is there any evidence of replacement of neural tissue by glia tissue as is the case in organic dementia or general paralysis. ($\times 85$)

which were observed in the two hospital cases and one asylum case that we recorded. The chromatolysis of the somatic cells may also be correlated with the slowness of utterance so characteristic of myxœdema. The marked chromatolytic changes which were observed in the cortex of the four asylum cases may be correlated with the mental confusion noted in these cases.

THE LIVING NERVE CELL AND CHROMATOLYSIS.

This basophile-staining substance which forms the Nissl granules does not exist as such in the living cells. If living cells are examined with the dark-ground microscope they are seen to be filled with small granules or globules, each of which after escaping from the cell remains discrete. They are refractile, and therefore appear bright white; this is due to a delicate covering film of a lipid substance which encloses a colloidal fluid probably consisting of a solution of salts and cell globulin (*vide* fig. 12). When the cell dies this colloidal fluid is coagulated and the precipitated proteid substance is massed together into little blocks—the Nissl granules. The film that covers each globule is stained by vital methylene blue, and a living cell stained by vital blue represents the appearance of an emulsion of minute faint blue globules. If the living cell thus stained be kept in an atmosphere of nitrogen the stored oxygen is used up and a leuco-base is formed, causing the globules to lose their colour, the cell being then stained a faint green. On admission of oxygen, the living cell again becomes blue. It thus appears that we have a large oxygen surface, like spongy platinum, within the osmotic membrane of the cell. When the cells die, the lipid film of the globulin-containing fluid is destroyed, coagulation occurs and the Nissl granules are formed. Examination of living cells of the spinal ganglia and spinal cord of animals in which the sciatic nerve has been cut shows that the cells have imbibed water. The refractile granules are not so densely packed, they seem to be pushed aside, and the nucleus becomes eccentric in position. It would be interesting to see what is the condition of the living nerve cells in experimental hypothyroidism, as we should then be able to see whether a similar condition occurs as is produced by section of the axon. The chromatolysis observed in hypothyroidism may be explained by a toxic condition of the blood altering the osmotic membrane of the nerve cells, and thus leading to imbibition of water; or it may be explained by the absence in the blood of thyro-iodine, or some substance essential for nerve cell metabolism. Probably both factors are a causative agency, but the absence of the gland secretion is the essential factor, because it is certain that the characteristic mental and physical symptoms and signs may greatly improve by treatment with administration of thyroid gland. The work of Edmunds, carried out in my laboratory, shows that thyro-parathyroidectomy causes a chromatolysis

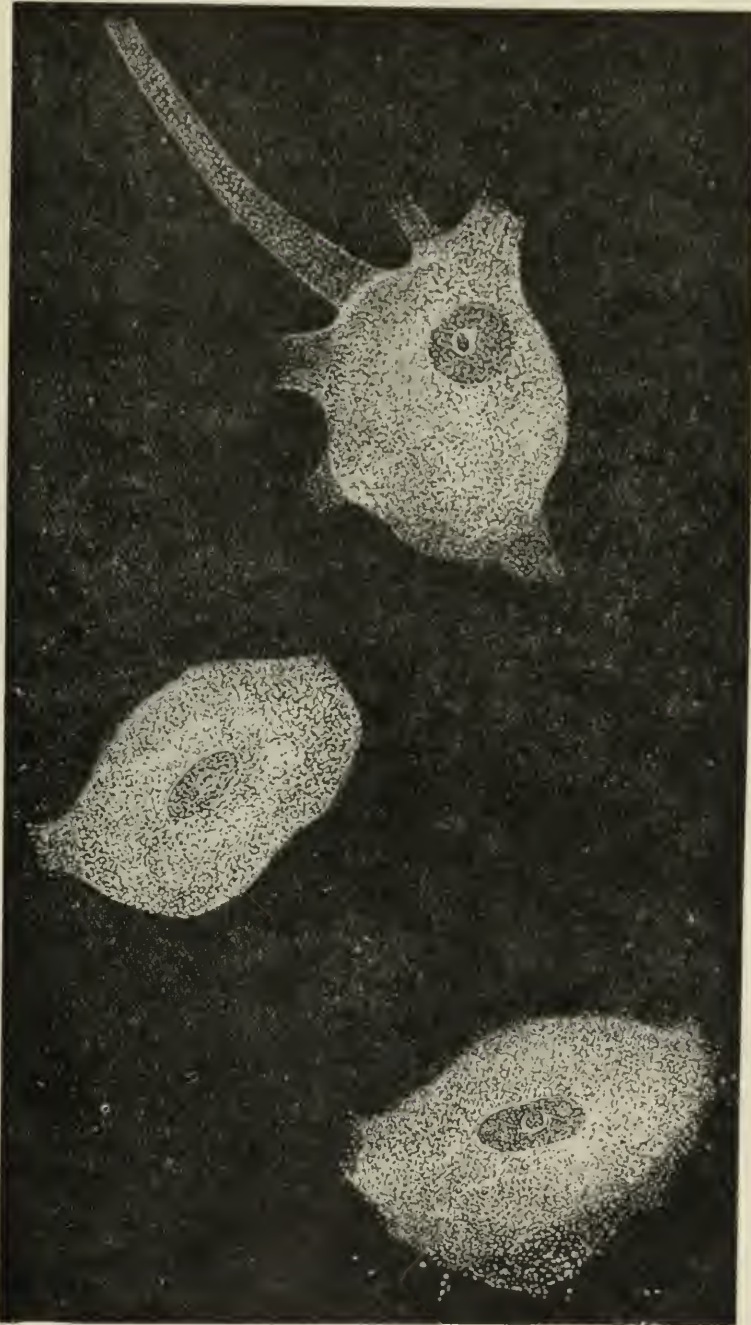


FIG. 12.

Drawing of an anterior horn cell (uppermost) with processes and two posterior spinal ganglion cells as seen by dark-ground illumination while still in the living state. (Obj. 4 mm., apochrom. oc. 4.)

of the nerve cells in dogs; his work also shows the importance of lime salts in prolonging the life of the animals.¹

Asylum cases frequently show but slight physical signs of myx-œdema, although the mental symptoms are marked, which affords an explanation as to why these cases have been erroneously diagnosed as alcoholic psychosis and dementia paralytica; consequently they have not been treated with the only drug that can do good. Cases should not be mistaken for general paralysis of the insane, for this disease can now always be diagnosed by the examination of the cerebrospinal fluid—an absence of lymphocytes and of a positive Wassermann reaction absolutely excludes general paralysis.

I have myself seen a case of agitated melancholia, or as it is often termed “manic-depressive insanity,” and heard of several cases, with few physical indications of hypothyroidism, which occurred in women about the climacterium, deriving great benefit from administration of thyroid gland. If my observations are correct, the treatment must not be delayed until destructive neuronie changes and substitutive glia hyperplasia have occurred. Whether we regard the mental syndrome as a consequence of a toxic condition or of a sub-minimal deficiency of an essential substance, or of the two combined, the fact remains that there is at the climacterium in women a group of mental symptoms associated with definite changes in the central nervous system and hypothyroidism.

¹ “The Changes in the Central Nervous System resulting from Thyro-parathyroidectomy,” *Proc. Roy. Soc. Med.*, 1912, v (Neur. Sect.), p. 179.

The Cerebral Mechanism of Speech.

(PRELIMINARY NOTE.)

By F. SANO, M.D.

THERE are a great number of methods employed in the study of the cerebral mechanism of language. One of the most important of them is the anatomo-pathological; it gives us the elementary facts as a foundation for further discussion, and each one of these facts may be an occasion of a new control for the theories of localisation as well as for the examination of the mutual influence exercised by the cortical areas, the lesions whereof cause defects in human speech.

I will demonstrate to you the brains of five patients who suffered from aphasia. And I take this early opportunity to express my thanks to the National Health Research Committee for the grant enabling me to work in the Pathological Laboratory at Claybury, with the permission of the Asylums Committee of the London County Council, and under the direction of Dr. F. W. Mott, who has kindly placed at my disposal the valuable material for clinico-anatomical research, a brief preliminary outline of which I will bring before your notice.

By means of exact and schematic drawings I will review each of the cases, of which I will show you the macroscopic specimens and the anatomical preparations, and I will begin with an extensive lesion of the left hemisphere, with its far-reaching influence on the whole cerebrospinal axis.

CASE I.

S. P., died aged 61. He must have had just before or in the first year after birth a destruction of the left *centrum ovale*, of which there remained only a hard and calcified tumour, which appears to be a tuberculoma, resulting in the formation of a large cyst with hemiatrophy of the left hemisphere. The external form of the hemisphere was not smaller than that of the right one, but the convolutions of the convexity, although of good appearance, are reduced to the grey matter, with a very thin remnant of white substance.

The lower parts of the frontal, temporal, and occipital lobes are the best preserved, in so far that a larger amount of white matter remains, but a considerable increase of neuroglia exists in this white substance adjacent to the temporal and occipital cortex.

The projection systems in every region except the frontal are absent.

The superior longitudinal bundle is completely destroyed. The internal association tracts are represented by a thin bundle representing the inferior longitudinal fasciculus; a tapetum exists in the lower parts of the ventricle where the ependyme persists on the temporal and occipital portions. In the lower third of the frontal lobe a layer of white matter of 2 cm. contains numerous association fibres. The cornu ammonis and the fornix suffered slightly. The olfactory nerve is smaller on the left side.

Both the lenticular and caudate nucleus of the left corpus striatum are but slightly reduced.

In its frontal part the corpus callosum is reduced to half its size, in its middle part to a thin layer; on the splenium a small bundle of fibres comes along the convolutions on the calcarine fissure, and so an indication of forceps is present. The anterior commissure is in a fairly good state.

Under these conditions it is remarkable to find a noticeable persistence of cell and fibre structure, indicating vitality in the grey matter forming the wall of the cyst. Although the large pyramidal cells are practically absent, we find in the third layer a number of middle-sized and small pyramidal cells, and a well-preserved striation wherever this ought to be.

It will be difficult to admit that the grey matter could have had any importance for a normal psychic activity; but it remained, living its own life, as a nervous tissue, active in the general process of metabolism and as an accumulator for discharge of energy with pathological short circuit. The patient had numerous epileptic fits.

The thalamus is reduced to a third of its normal size; its external nucleus is very atrophied. It is possible that in some way this consequence has been a direct one, produced by the internal pressure and the acute disease at the time of the destruction of the centrum ovale.

As a result of the hemiatrophy of the left hemisphere, the pyramidal tract is absent in its whole length, in both divisions direct and crossed.

It has been difficult to give an opinion about the red nucleus.

On the *left* side the posterior corpus quadrigeminum is atrophied, and also on the same side the inferior olivary body. This atrophy may be correlated with the change about to be described.

There is an atrophy of the whole of the right half of the cerebellum. Also on the right side the superior olivary body, the nuclei of the posterior columns in the medulla oblongata, and the column of Clarke in the spinal cord. The right half of the spinal cord is reduced in all its parts. Owing to the reduction of the anterior horn, I must especially call your attention to the fact that a careful examination has led to the conviction that the number of motor cells is very little decreased, and I venture to think that the reduction of the anterior horn is due more to the lessened number of association fibres and to a poor development of the processes of the ganglionic motor cells, than to a diminished number of these cells.

In the medulla oblongata there is also evidence of an equal development of the motor nuclei on the two sides.

For the general explanation of all these atrophies, local pathological conditions may be considered: also functional insufficiency has had its influence. S. P. was left-handed. He had been able to learn at school. He could read and write easily, and became a clerk. On admission, however, in 1897 (he was then aged 46), he was considered to be suffering from aphasia, and showed the symptoms by his inability to recall the names of common objects. He refused to accept a wrong word, and sometimes could complete the word when the first syllable was suggested. This kind of *amnésie de recollection* (Pitres) was the first indication of the beginning of slight progressive dementia.

CASE II.

A. C., at the age of 15, fell off a tram-car. He died at the age of 22. After his fall he had epileptic fits, and was later on operated upon for brain tumour. There was a right facial paresis; right hemianopsia, optic neuritis. No apraxia. The patient was unable to write with his right hand. His memory was defective, his speech slow, but the articulation fair (1913). Shortly before death there was a right hemi-paræsthesia and hemi-anæsthesia, right hemi-contraction, ptosis, and diplopia. The patient could still understand the questions; his articulation was poor, his speech slow and deliberate.

At the post-mortem examination the opening on the left temporal lobe, produced by the operation, reached the striate body. There was an extensive tumour in the temporo-occipital part of the hemisphere, and three small metastatic growths in the same hemisphere.

CASE III.

N. E. had a seizure when aged 56, and died at the age of 61. After the seizure her speech was very bad for some time: the words were badly articulated, or wrong words used (dysarthria and paraphasia). There was a slight facial paresis on the right side. She improved during the two years following the seizure. There remained some difficulties in utterance.

In the left hemisphere there is a softening in the precentral gyrus and in the beginning of the third frontal and the insula. The external capsule suffered slightly: the claustrum is preserved.

There is a patch of encephalitis in the *left* olivary body in the medulla oblongata, and the diseased artery which caused this has also produced pathological consequences extending as far as the *left hypoglossal nucleus*. The difficulties of utterance from which the patient suffered, and which were considered as consequences of her aphasia, might also have had this lesion for its origin. But this bulbar disease was not at all presumed during life, and would not have entered into consideration had not a complete examination of the lower centres been made.

CASE IV.

F. P., an epileptic since the age of 14, was suddenly affected with aphasia at the age of 50. He died at the age of 52. He spoke in monosyllables, slurring the words, and with difficulties in his articulation. He used wrong words (paraphasia), he could not write, nor could he copy. He was word-deaf. There was no paralysis.

At the post-mortem examination there was a softening in the left hemisphere, including the gyrus supra-marginalis, the gyrus angularis, the posterior third of the temporalis superior, and the middle third of the second temporal. At one place the softening reached to the ventricle, but as a general condition it was rather superficial, and the association bundles (superior and inferior longitudinal fasciculi) persist.

CASE V.

M. A. G., aged 63 at the onset of her illness: she died about two months later. The individual words were distinctly pronounced, but the patient jumbled her words; one could not make any sense of it. She understood the remarks, and answered "Yes" or "No"; sometimes she could say a short and elementary sentence. She was not deaf. She could read "Charing" and "word," but no more. Spontaneous articulatory speech. She could not repeat and could not write. It is not certain if there was apraxia or not.

A growth (small-celled sarcoma) was found in the temporal lobe, and a compression of the pedunculi, which had produced paraplegia and death.

The rapid review of these five cases, illustrating destruction of cortical regions connected with speech functions, lends support to the following conclusions:—

(1) Although the distinction between motor and sensory aphasia can be generally made, and is of importance for the problem of localisation, attention must be called to paraphasia occurring in cases of motor aphasia, and difficulties in utterance occurring in the cases of sensory aphasia.

(2) Age has a great influence upon the symptoms and the recovery of speech defects. Young individuals rapidly accommodate the right hemisphere to the loss of the left, so that the symptoms are less accentuated. Old individuals have greater difficulties in accommodation; in the latter, isolated lesions without general diseases of the arteries are exceptional, and so the symptoms are, as a rule, more extended, and recovery is more difficult.

(3) Disturbances in sensory centres are of larger and more definite influence than those in the motor areas.

1

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE EIGHTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1914-15

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1915

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SURGICAL SECTION.

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Surgical Section.

SUB-SECTION OF PROCTOLOGY.

November 11, 1914.

Mr. F. SWINFORD EDWARDS, F.R.C.S., President of the Sub-section,
in the Chair.

Case of Abdomino-perineal Excision of the Rectum for Carcinoma in a Woman, aged $76\frac{3}{4}$.

By ASLETT BALDWIN, F.R.C.S.

THIS patient was operated upon in May last and was aged $76\frac{3}{4}$ years at the time of operation. In addition to the most careful antiseptic technique, the area of the abdominal incision was infiltrated with 1 per cent. urea and quinine solution, also the extra-peritoneal tissues on each side. The areas of the internal incisions—the mesentery, between the bowel and the sacrum, the floor of the pelvis, and the ischio-rectal regions—were infiltrated with $\frac{1}{2}$ per cent. novocain solution, in order to block the nerves against transmitting shock impulses. During the whole operation saline solution was run into each side of the chest through two hollow needles. When the patient left the table she had a pulse of 84 and was in remarkably good condition. I think the excellent result in such an elderly patient was largely due to the nerve-blocking.

Mr. BALDWIN, in reply to the President (Mr. Swinford Edwards), said that urea and quinine solution was used for the sensitive abdominal wall because the anæsthesia lasted longer than with other local anæsthetics, in some cases up to several days. The internal incisions were not through such sensitive parts and did not cause after-pain from the sutures, so a more transient anæsthetic could be used, and he thought the weaker solution of novocain was less likely to interfere with healing.

Obscure Case of Œdema of the Sigmoid Mesocolon.

By IVOR BACK, F.R.C.S.

THESE are the notes of the case of a patient who was under my care at St. George's Hospital, and I am bringing them forward to-night in the hope that you may be able to throw some light on what remains to me rather a mystery.

The patient was a girl, aged 15. She was admitted to hospital on February 14 of this year. She had been quite well until one week before, when she suddenly passed about two pints of dark blood by the rectum. She did this daily up to the time of admission, and on more than one occasion the blood was seen by her doctor. The day before admission she vomited and complained for the first time of pain, in the lower abdomen on both sides.

When I first saw her she was not blanched and showed no obvious signs of having lost much blood. Her pulse was 120; her temperature 100° F. The whole lower abdomen was slightly tender and the left iliac fossa very slightly rigid. Rectal examination proved absolutely negative. I decided to watch her because the symptoms were vague.

The next day, at 10 a.m. she passed 3 oz. of bright red blood *per rectum*. At 2 p.m. she began to vomit. The abdominal pain became worse, mostly now in the left iliac fossa, and was paroxysmal in nature. The temperature dropped to 96° F. and the pulse to 100.

I opened the abdomen just to the left of the middle line below the umbilicus. Everything was normal except the pelvic colon, but here an extraordinary state of things was discovered. From the point where the descending colon becomes the sigmoid for a distance of 10 in. the mesocolon was extremely œdematous. It was so much thickened that the gut lay buried in it as if it were in a trough. The gut itself was unchanged except that the appendices epiploicæ were enlarged and œdematous. The surface of the mesocolon was glistening. The œdematous portion began and ended abruptly. Below the gut was normal. There was no peritonitis. I closed the wound.

Afterwards her temperature ran an irregular course and her pulse-rate remained high. The pain in the lower abdomen disappeared. Eleven days after the first operation the temperature rose suddenly to 104° F. and the pulse to 152. She now complained of pain in the right upper abdomen, and she was very tender over the gall-bladder.

I again opened the abdomen, this time to the right of the rectus, and found the gall-bladder much distended. It was full of what looked like London mud. There were no gall-stones. I put in a tube and drained it. I had the contents of the gall-bladder examined bacteriologically and a paratyphoid bacillus was grown. Dr. Hunt described it as a member of the typhosus and coli group, but certainly not distinctly belonging to either.

After the second operation she made an uneventful recovery and she is now quite well.

I suppose the two conditions were connected and both were due to this paratyphoid bacillus. But I am quite unable to explain how the sigmoid mesocolon could become so œdematous without apparent change in the gut. If it had been a thrombosis I should have expected gangrene to have followed.

Case of Inoperable Carcinoma of the Rectum becoming Operable under Radium Treatment.

By W. SAMPSON HANDLEY, M.S.

MR. J. C., aged 60, had for the past eighteen months experienced rectal irritation. The bowels were open three or four times each morning, with a slight trace of blood. The symptoms were supposed at first to be due to a small polypus which was detected just inside the internal sphincter. An anæsthetic was suggested by his medical man, Dr. Neville Spriggs, in order to make a complete examination of the rectum, but the patient objected to this and did not place himself definitely under treatment.

A year after the first symptoms there was no loss of weight; a small polypus was still present, but the symptoms had abated. Shortly afterwards Dr. Spriggs found with the proctoscope a small bleeding area, which, however, was soft to palpation, situated about 5 in. above the anus. Irritation and slight bleeding returned, and Sir A. Bowlby pronounced the condition to be an inoperable carcinoma.

On August 22 the patient was brought to see me. On the left side of the rectum, in the situation of the recto-vesical fold, a rather fixed submucous thickening could be felt. An anæsthetic was given and with the sigmoidoscope an ulcer was found 13 cm. from the anus. The instrument could not be passed through so as to see the upper

4 Handley: *Inoperable Carcinoma becoming Operable*

edge of the ulcer. Portions of its lower edge were removed through the sigmoidoscope, were examined by Dr. Ernest Shaw, and were found to be carcinomatous. I agreed that the condition was inoperable and advised radium treatment.

Dr. Finzi was asked to see the case, and early in September, using the sigmoidoscope, he applied 155 mg. of radium bromide to the ulcer, passing it into a stricture which he detected higher up above the lower

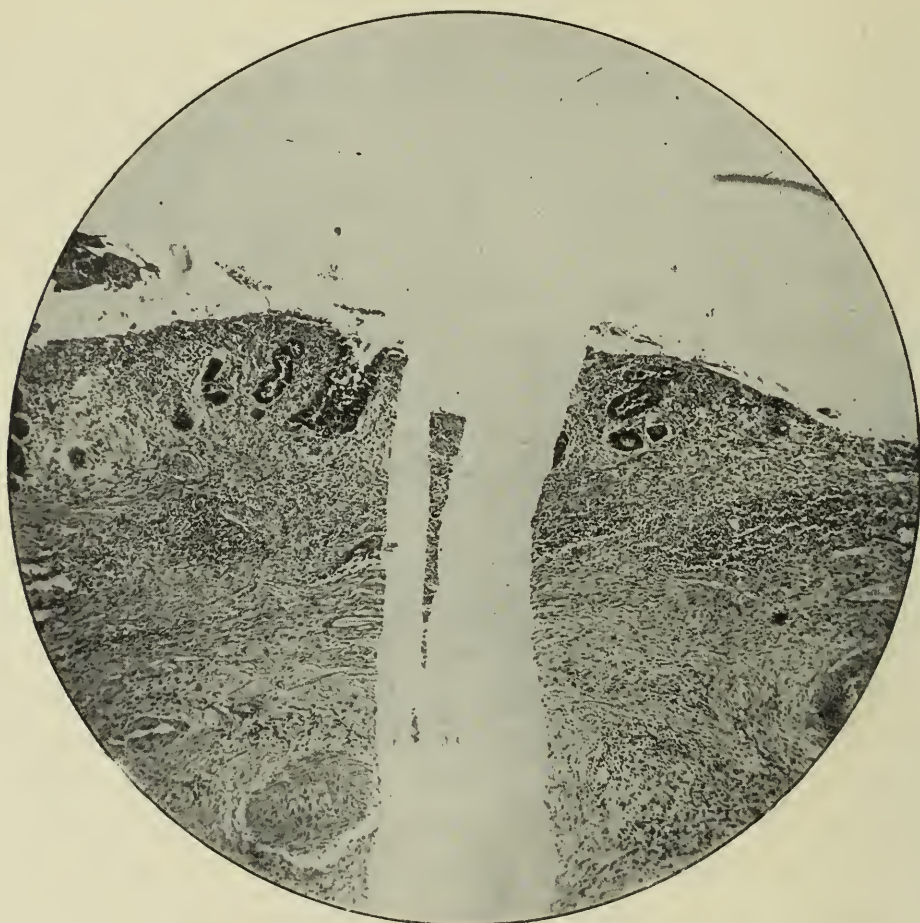


FIG. 1.

The edge of the malignant ulcer after radium treatment, seen under a low power. Note on the left the remainder of the normal crypts of Lieberkühn. Note also the flatness of the edge of the ulcer and the fibrotic character of the growth beneath. The vertical cleft seen in the middle of the figure was made to orientate the section.

edge of the ulcer, and leaving it there for twenty-four hours. The tube was then pulled down and left opposite the ulcer for twenty-four hours. A fortnight after the application there was evidence of a sharp reaction, with considerable irritation of the bowel, tenesmus, diarrhoea,

and pain in the back which required treatment by rest in bed with small doses of nepenthe. The reaction passed off, and from the thirty-fifth day onwards the bleeding and pain ceased, and the patient found himself better altogether, and able to walk more briskly and freely than before the radium application. On rectal examination the edges of the ulcer were flat, and the stricture appeared to be a simple stricture.

He came up with a view to a further application of radium on



FIG. 2.

Taken from the middle of the growth. Shows under higher power the fibrotic changes occurring in the growth, with atrophy and partial destruction of the cancer cells, and dilatation of some of the capillaries.

October 22, and marked local improvement was found. Since it appeared improbable that radium would produce a radical cure, it was decided to take advantage of this improvement to attempt a radical operation. If this proved to be impossible a colostomy was to be done, and the colostomy opening utilised for radium treatment

of the growth from above, possibly to be carried out by burying radium in the growth.

On October 30 the abdomen was opened in the middle line and the growth appeared to be removable. The sigmoid was clamped and divided with thermo-cautery; its divided ends were then closed by basting stitches. The upper end was brought out by a stab incision in the left rectus and the whole of the bowel was removed by the usual abdomino-perineal operation. The peritoneum on the floor of the recto-vesical pouch looked somewhat suspicious, and was removed as freely as possible consistent with the subsequent repair of the pelvic floor. The bladder was to some extent utilised to restore the pelvic floor. The ureters were not seen. Owing to the large size of the growth it was not possible to push the rectum down beneath the pelvic floor to repair the latter. The operation had therefore to be conducted in three stages—(1) abdominal, (2) perineal, (3) abdominal—and was consequently a prolonged one. Radium, protected by gauze, was left in the pelvis, the gauze projecting from the lower part of the abdominal wound. Two tubes of radium were also introduced into the pelvic wound. At the close of the operation the patient was in excellent condition with a pulse of almost normal rate. The radium was left in position for twenty-four hours, and the administration of gas was necessary for its removal. It is possible this interference predisposed to sepsis; the fact remains that a large pelvic abscess formed on the right side about the tenth day and had to be opened under gas. A smaller one formed on the left side of the pelvis and was opened a week later. There was also some suppuration in the abdominal wound and in the pelvis. Prior to the opening of the first abscess the patient's condition seemed very grave, his pulse being about 130, although at no time was there any sign of intestinal obstruction, and the bowels acted freely. He left the home with the abdominal wound soundly healed on December 16, about seven weeks after the operation, being then able to walk and to ascend a flight of stairs. The day before he left the home 150 mg. of radium were left in the pelvic sinus as high up as possible for twenty hours.

Subsequent to his return home he had an alarming attack of acute pyelo-cystitis, but since his recovery from this attack he has remained in good health up to the present time, a period of over a year.

Dr. N. FINZI said that the first radium application was made on September 6, 1913, by means of a sigmoidoscope. The radium was passed farther than a large mass in the left side of the bowel into a stricture, and 150 mg. of

radium bromide, surrounded by a sheath of 2 mm. of platinum, introduced at the end of a long flexible silver rod was left nine hours in this stricture, when it was found possible to push it higher up, and in this higher situation it was left twelve hours, and it was then withdrawn lower than the original situation and left for ten hours longer. When next seen it was found, as Mr. Handley has described, that the growth was considerably smaller, and it was decided that the best method of treatment would be by operation, which was performed on October 30, 1913. 160 mg. of radium bromide surrounded by shields of a thickness varying from 1 mm. of silver to 2 mm. of platinum, and the whole surrounded on all sides by about 2 cm. of gauze, were left in the abdominal wound for twenty hours, and 50 mg. of radium bromide, screened by $\frac{1}{2}$ mm. of silver and 1 mm. of lead and 4 mm. of gauze, were left in the perineal wound for the same period. On December 15, 1913, 160 mg. of radium bromide with shields similar to those previously used were placed at the apex of the perineal sinus. With regard to the suppuration which occurred, he had heard of this in an abdomino-perineal operation where no radium had been used and did not think it could be entirely attributed to this cause. As to the general results obtained by radium treatment in rectal cases, they varied considerably. Naturally the cases submitted for treatment were usually very far advanced. The only early case which he had had was quite recently, and was a small carcinomatous ulcer just inside the anus, in a patient whose general condition precluded operation; this had healed quite satisfactorily with three applications of filtered radium rays, but whether the result would be permanent it was impossible to say. He had another case of a carcinoma on the front wall of the rectum which was on the border-line of operability if the patient had been strong, but he had severe cardiac disease and operation was quite out of the question. This patient was well, as far as his rectum was concerned, this spring, four years after the commencement of the treatment. Of course, there were a number of cases in which the treatment either failed or only gave relief. In his experience the rapidly growing cases were more favourable than the more slowly growing ones, and as an instance he might mention a case that he had had early this year, whom he saw one Thursday evening with a recurrent carcinoma that had rapidly followed an operation: the ulcer was just inside the rectum and, when first seen, reached to the middle line posteriorly and about halfway across the left side of the rectum, the diameter of the ulcer being about $1\frac{1}{2}$ in. It was decided to apply the radium on the Saturday, forty-eight hours afterwards, and when this came to be done it was found that the ulcer had extended across on to the right side of the bowel, reaching about halfway across, and on the left side it nearly reached the front. In the forty-eight hours it had at least doubled its size. Another point of recurrence which lay external to the anus and quite separate from it had, during the same period, extended up to the mucous membrane. The application of radium in this case had been followed by a result which was so far extremely satisfactory. Vigorous treatment had been undertaken, with the result that a certain amount of radium ulceration had been produced, but there was now no sign of the

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original disease and the radium ulceration was steadily improving. These rectal cases were liable to be followed by a considerable amount of cicatricial contraction when they were successful, and they must be kept dilated with bougies. Another point worthy of notice was that the rectal mucous membrane was extremely sensitive to radium rays, and every effort must be made to keep the radium well away from the healthy parts of the rectum. What influenced them in the decision to treat this particular case by operation was the difficulty of getting the radium to the growth in such a way as to make certain that they had treated the whole of it thoroughly.

Injuries to the Bowel from Shell and Bullet Wounds.

By P. LOCKHART MUMMERY, F.R.C.S.

INJURIES to the rectum or pelvic colon which occur in war, whether from bullet or shell wounds, must of necessity be very serious; and as a fact, the statistics of previous wars show that injuries of this nature are attended by a higher mortality than injuries to other parts of the alimentary canal. Many of these cases must be fatal owing to concomitant injuries of the large vessels in the pelvis, or to resulting peritonitis. In the Boer War the mortality from wounds of the rectum was 30·7 per cent. of those so wounded, death being due to peritonitis, internal hæmorrhage, or septicæmia. We have no means yet of ascertaining the percentage of mortality from such wounds in the present war, but already there must have been a number of such cases of wounds involving the large bowel under treatment in our home hospitals. It is to be hoped that, as the result of increased knowledge and improved technique, we shall be able during the present war to bring the percentage of mortality for wounds of this nature down to a much lower figure than in previous campaigns.

As these wounds present unusual difficulties and require special treatment, it does not seem out of place to discuss them at this early stage of the war, if by so doing we can give any assistance to ourselves and other surgeons who may have to deal with such cases during the continuance of the war. In my position as Surgeon to King Edward VII's Hospital for Officers I have seen several of these cases during the last three months.

The injuries vary from clean bullet wounds to large, septic, lacerated wounds due to fragments of shell. There can be no doubt that a high-velocity Mauser bullet may pass straight through the abdomen, or across

the pelvis, penetrating the large and small bowel, without causing a fatal, or even a serious result, always provided that certain conditions are present. Of these conditions the chief are: The bullet must be travelling at a relatively high velocity, the intestines must be more or less empty of fluid contents, and the proper first-aid treatment must be administered. The Boer War taught us that, in the case of penetrating wounds of the abdomen, the best results were obtained by giving complete rest to the intestines for forty-eight hours after the injury, and that with a view to obtaining these conditions treatment should be confined to giving morphia in full doses, withholding all food, and providing as much rest as is compatible with the circumstances. It was found that such treatment allowed the small wounds in the bowel to seal themselves off, and prevented the risk of faecal leakage.

I should here like to express my unqualified admiration of those heroic members of our profession who, often at the risk of their lives, are so ably administering first-aid to the wounded behind the firing line. I have now seen a considerable number of our wounded on their arrival in London, and have been much struck by the efficient treatment which they have received on the field. The good results which, I believe, are being obtained in the treatment of our wounded soldiers depend above all upon the efficiency of the medical treatment at the first dressing station.

The following case which has just recently come under my care affords a good example of the comparative harmlessness of a clean bullet wound involving the bowel. The patient was shot through the pelvis, the bullet entering at the external abdominal ring on the left side and passing out through the middle of the right buttock. Fortunately the patient had urinated only a few minutes before he was struck, and consequently the bladder was not injured. The bullet must, however, have passed through the small intestines in the pelvis, and almost certainly through the upper part of the rectum. It emerged close to the sciatic nerve on the right side. The wound was kept aseptic, and the only inconvenience from which the patient has suffered so far has been severe neuritis in the sciatic nerve, with temporary paralysis of some of the muscles. There have been no bowel symptoms at all.

Wounds involving the large bowel are generally complicated by other injuries, such as fractures of the pelvis, injury to the bladder, or damage to the large nerve trunks. One or more of the nerve trunks may be cut, but more commonly they are only concussed. The

symptoms in either case are, at first, the same—namely, motor and sensory paralysis, generally only partial. When the nerve is only concussed the symptoms begin to pass off in a few days, although it may be some weeks before there is complete recovery. I have seen two cases in which the sciatic nerve was concussed just at its point of exit from the pelvis. In both cases there was temporary complete loss of movement, followed by rapid recovery, but also by severe neuritis which lasted for some time. One officer shot through the pelvis had apparently some injury of the sacral plexus. There was acute hyperæsthesia of the skin in the anal region, with resulting pain and tenesmus on defæcation, and retention of urine. These symptoms, however, passed off in a few days.

The most difficult cases are those associated with fracture of the pelvis and a septic wound. One officer under my care was shot through the pelvis, the bullet entering at the symphysis and emerging close to the sciatic notch, between it and the acetabulum, on the right side. The pelvis was extensively cracked, and the wound had become septic before the patient was admitted to the hospital. When the right leg was moved the sensation was rather like that of moving the parts of a broken basin. The bullet in this case had passed close to the rectum, although without injuring the mucous coat. With a finger in the rectum one could easily distinguish the track of the bullet as a firm elastic ridge passing along the left side of the bowel. Fortunately no fæcal fistula had developed. The case was treated by free drainage with tubes, and fixation of the patient in a Chappell splint. Healing in such cases is likely, I am afraid, to be very slow, as considerable portions of bone must separate before healing can occur, and this is a long and tedious process which cannot well be hastened by surgical means.

The worst cases are those in which a shell wound is complicated by a fæcal fistula and fracture of the pelvis. The following case is one of the most serious that I have met with yet. The officer in question was wounded at the battle of the Aisne by a shrapnel shell. There was very severe hæmorrhage on the battlefield, and three days after he was injured a fæcal fistula developed. On admission to the hospital there was a large lacerated wound on the left side above the hip-joint. This led down to a hole about the size of a two-shilling piece, through the wing of the left ilium, and all his fæces were being discharged through this opening. The patient was profoundly septic, and a secondary hæmorrhage occurred soon after his admission. As it was

impossible to deal with the wound while it was complicated by a faecal fistula, I decided to open the abdomen, and either to perform a temporary colotomy or, if possible, to close the wound in the bowel. On opening the abdomen I found that the sigmoid flexure had been injured on its mesenteric border, and that it was adherent to the inside of the ilium. I succeeded in separating it, and after trimming up the edges I closed the wound in the bowel with sutures. The abdomen was closed and the wound drained through a hole in the ilium, another opening being made above the ilium, in the loin, through which drainage-tubes were introduced into a large lacerated wound in the posterior abdominal muscles where fragments of the shell had buried themselves. After this it was possible to deal properly with the wound. There was no further trouble from the faecal fistula, the bowels acting in the normal manner. The wound was exceedingly septic, as one had to deal with a compound septic fracture of the ilium complicated by *Bacillus coli* infection. A rather alarming secondary hæmorrhage from one of the lumbar arteries was, however, the only complication which occurred, and the patient, I am glad to say, is now making an excellent recovery.

In this case I was able to close the wound in the sigmoid successfully, and so to save the patient the discomfort of colotomy. But this must often be impossible, and I believe that in most cases the best way to deal with severe wounds complicated by a faecal fistula is to perform a temporary transverse colotomy, at the same time thoroughly opening up the wound and providing free drainage. After the wound in the bowel has healed the colotomy can always be got rid of by a secondary operation. It is obviously impossible to deal satisfactorily with a serious wound of the pelvis if it is complicated by a faecal fistula, and I feel sure that the best treatment in such cases is a temporary colotomy. This must, of course, be done in such a way as to leave a good spur, so that there is no possibility of faeces passing on into the distal bowel. The old-fashioned colotomy would be quite inadequate to deal with such cases, and the best way of performing colotomy in these cases is by means of a glass rod placed under the bowel. The performance of a colotomy will, I think, be the best routine treatment in all cases where there is a large or serious wound complicated by a faecal fistula into the rectum or pelvic colon.

In the case of a wound which is quite small and is not complicated by fractured bone, but is complicated by faecal leakage, an attempt may, I think, justifiably be made to get it to heal without resorting to a

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colotomy, provided that the patient has not developed a serious degree of sepsis. If such an attempt is to be made the rectum should be drained by means of a tube introduced through the anus. Unfortunately, however, few patients can tolerate the presence of a tube through the anus for long, unless the sphincters have been divided. In the case of large lacerated wounds of the bowel temporary colotomy is indicated as soon as possible. The closing of the colotomy is not a very easy matter, but, after all, this can be postponed until the patient is in the best possible circumstances as regards hospital accommodation and skill.

Finally, I should like to reiterate the view which I hold most firmly, that wounds involving the bowel should not be treated by immediate operation, even if proper surgical facilities are available. I believe that very few exceptions should be made to this rule, and that the proper treatment is, almost invariably, complete rest, absence of food, and the administration of morphia; surgical treatment should be reserved till later for the treatment of secondary complications.

Mr. SAMPSON HANDLEY said that, although there was general agreement as to the possibility of expectant treatment in gunshot injuries of the abdomen in war conditions, he could not subscribe to Mr. Mummery's extension of the same doctrine to gunshot wounds in civil practice, where intervention could be practised under ideal conditions within a short time of the accident. Under peace conditions he considered that immediate laparotomy was the right treatment for penetrating wounds of the abdomen as soon as the stage of profound shock had passed off. The danger of waiting was well illustrated in a case which came under his observation of a small boy who shot himself in the right iliac fossa with a toy pistol. Alleging toothache, he went to bed, thus putting himself under favourable conditions for localisation of the injury and the prevention of extravasation. Thirty-six hours later he was brought up to the hospital with obvious general peritonitis, and death occurred in spite of operation. Although few opportunities occurred in civil life, he was able to quote a case of successful laparotomy for a revolver wound in the region of the umbilicus in a young man. The abdomen was immediately opened, and the small intestine was rapidly examined from the ileo-cæcal valve to the duodeno-jejunal flexure. Five bullet holes were found and were closed by purse-string sutures. An examination of the large intestine disclosed a single hole at the lower edge of the transverse mesocolon. In the posterior parietal peritoneum a freely bleeding bullet aperture was found and was sewn up. The patient made an uninterrupted recovery. While it was most necessary to emphasise the importance of conservatism in the treatment of these injuries during the war, the doctrine could not be extended to peace conditions.

Surgical Section.

January 6, 1915.

Mr. C. A. BALLANCE, M.V.O., Vice-President of the Section,
in the Chair.

Pneumo-peritoneum of Three Days' Duration following Resection of a Carcinomatous Rectum, and probably induced by the *Bacillus aerogenes capsulatus*; Evacuation of the Gas and of a Pelvic Abscess; Recovery; no Recurrence after Five Years.

By JOHN D. MALCOLM, F.R.C.S.Edin.

IN the following paper the clinical history of an unusual, if not unique, case is recorded, and an attempt has been made to summarise our knowledge of the aerogenic bacilli which from time to time produce such extraordinary effects, for the most part of a very dangerous character.

A woman, aged 60, was admitted to the Samaritan Free Hospital on account of repeated hæmorrhages from the bowel, accompanied by pain, if she became constipated, and loss of weight, these symptoms having been noticed within the previous six months. In the posterior wall of her rectum there was an ulcerated tumour, as shown in the figure, the lower edge of which was about 3 in. from the anus. It caused no fixation of the parts. Otherwise the patient's health was good, and she was decidedly fat. Her mother died of mammary cancer.

On December 31, 1909, the rectum was removed by the combined abdominal and perineal method described by C. H. Mayo.¹ Two very soft glands, an inch long, symmetrically placed, at the level of the pelvic brim, were removed, and subsequent examination showed that

¹ *Surg., Gyn., and Obstet.*, 1906, iii, p. 236.

they consisted chiefly of fat cells, no malignant growth being found in them. No other glands were felt. The bowel was divided about 6 in. above the disease, and its cut ends were inverted and sutured. The lower end of the sigmoid was mobilised, and the rectum, with as much fat as possible, was separated and everted through the anal canal. The patient was then placed in the lithotomy position and the rectum was divided between the disease and the internal sphincter, which last was left intact. The lower cut end of the rectum was retained outside the anus by forceps, and through it the end of the sigmoid flexure was drawn down. The sutures closing the sigmoid flexure were removed, and the ends of the bowel were adjusted and fixed together by interrupted sutures. The parts were then replaced in the pelvis, and thus the continuity of the intestine was restored, its sphincters being intact. The apposition of the ends of the bowel was so exact that Mayo's recommendation to introduce a drainage-tube between the coccyx and the anus was not followed.

After the operation the patient showed no unfavourable sign during the first five days, the highest temperature being 100° F., and the highest pulse-rate 112. On the third day gases escaped through the anus, and on the fifth the bowels moved in response to a saline purgative. On the sixth morning the abdomen was enlarged; it was resonant all over, and the distension was supposed to be intestinal. The abdomen became increasingly hard and tense both externally and on vaginal examination, and yet there was no vomiting, and the bowels never failed to move when a purgative was given. On the ninth day the highest temperature was 99·2° F. The pulse-rate was rising, but only 118, and of fairly good volume; respiration was not greatly distressed, and the general condition of the patient was not alarming, but in view of the extreme and increasing distension, it did not seem that expectant treatment could be safely continued. The indications of a pneumo-peritoneum were clearly present, but I did not know that this could occur in a patient otherwise so well, and the condition was not recognised until the abdomen was reopened.

An incision was made far back in the right loin with the intention of opening the ascending colon or showing that it was not distended. When the muscular wall of the abdomen and some fat were divided, a thin membrane bulged into the incision, and, on pricking it, gas escaped with a whizzing sound, but without odour. At the same time the abdomen softened and diminished in size. All doubt as to the nature of the gas-containing cavity was removed by the appearance of a piece

of omentum through the wound. The incision was plugged, and the recent scar in the middle line was reopened. As this was done the abdominal wall flopped down on the intestines, which were retracted, and occupied a quite small space at the back of the peritoneal sac. A considerable quantity of gas necessarily escaped, and Dr. R. D. Maxwell, who was assisting, confirmed my observation that this gas was odourless. The peritoneal cavity did not contain any fluid beyond its ordinary moisture, and its surface showed no sign of irritation, being, on the contrary, very pale. The intestines were not adherent except in the pelvis, where all the parts were matted together. On separating these, a large amount of very offensive pus was released, and the abscess cavity which contained no free gas was drained through the lower end of the median incision, the rest of which was closed, and healed by first intention. No further abnormal accumulation of gas took place. The bowels moved on the fifth day after this second operation, and the highest temperature after it was 100° F. The small loin wound was drained for a few days and healed quickly, but with some slight superficial suppuration. The pelvic abscess, which never discharged any faecal matter, was complicated by a secondary deep-seated collection of pus on each side. That on the left, which appeared first, opened into the central abscess, and with it healed after a short time. The collection on the right side was opened later by an incision in the right groin, and this part healed very slowly—chiefly, I think, because the patient felt quite well, and would not keep quiet. She left the hospital on June 1, 1910, with all her wounds firmly united, and her condition was then in every way satisfactory, except that the line of anastomosis of the bowel was fixed by cicatricial tissue. Although this tissue was unyielding, there was abundant room for the passage of solid fæces.

The patient, now aged 65, has persistently refused to see any medical man since she left the hospital, but, on December 28, 1914, five years after the operation, a report was obtained from her daughter that her general health was good. Her only trouble is constipation, for which she takes senna pods every second day. At times there has been some yellow discharge in the stools, but never any blood, and now there is no discharge.

The specimen removed was preserved by Dr. Cuthbert Lockyer, and the figure shows the growth very well. Dr. Lockyer described it as a glandular carcinoma. An ample margin of healthy tissue was removed above the tumour. Below it the excision was not so free, but a full

$\frac{1}{2}$ in. of healthy-looking tissue was seen beyond the growth at this part when the specimen was fresh.

The absence of evidence of recurrence five years after removal of the growth is satisfactory, but the pneumo-peritoneum constitutes the chief interest of this case. It is inconceivable that gas could escape into the peritoneal cavity from the intestine during three days through an opening, however small, without an escape also of organisms which would induce a diffuse general peritonitis, of which there was no sign. It seems quite impossible also that gas could escape from an abscess in which there was no gas.

It would appear, therefore, either that the gas passed through the wall of the healthy collapsed intestine into the peritoneal cavity in opposition to an increasing pressure, or that the gas developed within that cavity. But there is no evidence that gas ever escapes through a healthy intestinal wall, and therefore the only explanation of the condition found that seems at all feasible is that the gas developed in the peritoneal sac. The possibility that this may be the correct solution is beyond dispute.

Many descriptions of aerogenic organisms have been published, but only two are generally recognised as acting upon the tissues apart from putrefactive changes. These are the *Bacillus aerogenes capsulatus* of Welch and the *Bacillus œdematis aerobicus* of Sanfelice.

The *Bacillus aerogenes capsulatus* was isolated by Welch in 1891, and described in a most illuminating paper¹ by Welch and Nuttall in 1892. Eugene F. McCampbell, writing in 1909,² said that *Bacillus enteritidis sporogenes* (Klein), *Bacillus perfringens* (Veillon and Zuber), *Granulo-bacillus saccharobutyricus immobilis* (Schattenfroh and Grassberger), *Bacillus anaerobicus cryptobutyricus* (Achalme), *Bacillus cadaveris butyricus* (Buday), *Bacillus* of Cesaris Demel, and *Bacillus emphysematis vaginæ* (Lindenthal), are undoubtedly *Bacillus Welchii*.

Welch and Nuttall reported the case of a syphilitic phthisical mulatto man, a patient under the care of Professor (now Sir Wm.) Osler. Death occurred suddenly after repeated hæmorrhages, but not during a hæmorrhage, from a rupture of an aortic aneurysm through the chest wall. No gas was observed in the tissues before death, but

¹ "A Gas-producing Bacillus (*Bacillus aerogenes capsulatus*, nov. spec.), capable of Rapid Development in the Blood-vessels after Death," *Bull. Johns Hopkins Hosp.*, July-August, 1892, p. 81.

² "The Toxic and Antigenic Properties of *Bacterium Welchii*," by Eugene F. McCampbell, *Journ. of Infect. Dis.*, 1909, vi, pp. 537-563.

after eight hours, in cool weather, there was a diffuse swelling with emphysematous crackling, especially marked over the lower part of the neck. Gas-bubbles were found in many arteries and veins and in almost all the tissues. The gas ignited with a slight detonation and burned with a pale blue flame. An analysis of the gas from another case showed: Hydrogen, 64·3 per cent.; carbon dioxide, 27·8 per cent.; nitrogen, &c., 8·1 per cent.¹ It had no distinctive odour and there was no putrefactive odour or green discoloration of the body.



Rectum and tumour viewed from upper end of bowel.

Wherever the bubbles were found there was a bacillus which was easily seen in coverslip preparations. Aerobic cultures remained sterile. Anaerobic cultures yielded numerous colonies of the bacillus seen in the fresh tissues and no other species developed.

An injection of $2\frac{1}{2}$ c.c. of a culture of this organism into a vein of

¹ E. K. Dunham, *Bull. Johns Hopkins Hosp.*, 1897, viii, p. 69.

a rabbit produced no symptoms in five out of six experiments. In the sixth, death followed after twenty-one hours, the animal having been seen quite lively half an hour before it was found dead, and death was followed by an enormous development of gas in the tissues. This rabbit was pregnant and two of her young were macerated and decidedly smaller than the others and had apparently been dead some time. After the same injection into a vein, if the animal was killed within two days there followed all the phenomena of gas development observed in the man from whose tissues the bacillus was obtained. The development took place with "almost startling rapidity" if the rabbit was killed a few minutes after the injection was made and kept at a temperature between 30° C. and 35° C. When a culture of the bacillus was injected into a blood-vessel of a rabbit after death bubble formation spread slowly and to a limited extent along the vessel.

The evidence was conclusive that the gas-producing bacillus circulated in the blood and was harmless in the dose injected into the vein of a healthy rabbit, but that it retained its vitality for at least forty-eight hours and was able to grow vigorously if local or general death of the tissues occurred within that time.

Welch and Nuttall pointed out that many cases in which it had been supposed that death was caused by the entrance of air into a vein, particularly many puerperal cases of this kind, were probably examples of a development of gas caused by an aerogenic bacillus.

In the year following Welch and Nuttall's publication, E. Fraenkel described a bacillus as the cause of emphysematous gangrene, which he named the *Bacillus phlegmones emphysematosæ*.¹ He studied the results of introducing the bacillus, not into the blood-stream, but into the subcutaneous tissues, and found no effects from the inoculation of mice. Sometimes rabbits showed local œdema with necrosis of the skin followed by recovery. But in guinea-pigs the effect produced was essentially the same as the condition of malignant œdema in man, that is to say, there was a spreading gangrene, gas formed in the tissues, and death followed. There was no suppuration unless pyogenic bacteria were also introduced. The bacilli were usually localised in the necrotic area and did not affect the organs generally even after death.

In 1896, Welch and Flexner² stated their belief that the *Bacillus phlegmones emphysematosæ* of Fraenkel was identical with the *Bacillus*

¹ *Centralbl. f. Bakteriolog.*, Jena, 1893, xiii, p. 13.

² *Journ. of Exper. Med.*, New York, 1896, i, p. 1.

aerogenes capsulatus of Welch, and added that Fraenkel had assented to this view in a private communication to one of them. In their paper Welch and Flexner recorded twenty-three cases of gas in the tissues, in which with one exception the *Bacillus aerogenes capsulatus* was identified by the characteristic development of gas which took place if a culture was injected into the blood-stream of a rabbit, and it was killed a few minutes later. There was not a single recovery in this series of cases unless the part affected was amputated. In some cases the gas accumulated in the peritoneal or pleural cavity without external wound and without wound of the intestine or lung, but never without hæmorrhagic infarction of an adjacent viscus. In one case there were small gas cysts involving all the tissues of the intestine in two well-defined areas. The *Bacillus aerogenes capsulatus* was found in connexion with these gas cysts, but in no other part of the body nor in the blood-stream. It was found in the intestinal contents.

A case recorded by Graham, Steward and Baldwin¹ was quoted, in which a woman, aged 35, after a miscarriage which was possibly induced artificially, became emphysematous all over her body four hours before death. This occurred fourteen hours after a prolonged rigor which was the first symptom. After death the body became twice its normal size. The case shows that a development of gas may be induced throughout the tissues during life, but this is a very unusual phenomenon, although in the liver the tissues have been found altered and degenerated with disappearance of the nuclei around masses of the bacilli, and these changes have been regarded as an indication that the bacilli grow in the living tissues.

The *Bacillus œdematis aerobicus*, which is also concerned in a formation of gas in the tissues, associated with a spreading gangrene, was isolated and described by Sanfelice.² It would appear that this and the *Bacillus aerogenes capsulatus* cannot at present be certainly distinguished except by an exhaustive bacteriological examination of the organisms in the affected tissues.

Corner and Singer³ published a case in which they attributed the gas formation to an aerobic organism "which is very closely allied to

¹ *Columbus Med. Journ.*, 1893, quoted by Welch and Flexner.

² *Annali del Istituto d'Igiene Sperim. dell' Univ. di Roma*, 1892, n.s. i, p. 365. Quoted by Dudgeon and Sargent.

³ "Emphysematous Gangrene," Edred M. Corner and H. Douglas Singer, *Trans. Path. Soc. Lond.*, 1901, lii, pp. 42-60.

the members of the group of colon bacilli,"¹ and which they believe to be identical with the *Bacillus œdematis aerobius* of Sanfelice.²

Dudgeon and Sargent³ also published a case and went very fully into the bacteriological characteristics of the bacillus isolated. They applied Welch's crucial test for the differentiation of the *Bacillus aerogenes capsulatus*—namely, the introduction of a pure culture into the vein of a rabbit which was killed a few minutes later—and they did not observe the development of gas after death which is caused by the *Bacillus aerogenes capsulatus* in such circumstances. They concluded that their case and that of Corner and Singer were "examples of that small group of cases of emphysematous gangrene which are caused by the *Bacillus coli*."⁴

Although a bacillus was isolated which induced gas formation in various media, Dudgeon and Sargent pointed out, as Professor Welch did before, that with this bacillus no one has yet succeeded in producing gaseous phlegmons in the tissues of the various inoculated animals.⁵ But gas formation has been induced by introducing a pure culture of this bacillus into the tissues with other bacilli, such as the *Staphylococcus pyogenes aureus* (Chavigny) and the *Proteus vulgaris* (Muscatello and Gangitano).⁶ The circumstances in which the *Bacillus œdematis aerobius* causes gas formation in the tissues are not so definitely worked out as are those in which the *Bacillus aerogenes capsulatus* does so. The former is rarely, if ever, found in wounds in pure culture.

I do not propose to set down here the characteristics of the two bacilli originally described by Welch and Sanfelice. These are fully detailed in the various papers by Sanfelice, by Welch and his colleagues, by Corner and Singer, and by Dudgeon and Sargent, in which numerous references to the works of others will be found.⁷

¹ Loc. cit., p. 43.

² Loc. cit., p. 45.

³ "Case of Emphysematous Gangrene, caused by the Colon Bacillus," by Leonard S. Dudgeon and Percy W. G. Sargent, *Trans. Path. Soc. Lond.*, 1904, lv, pp. 107-118.

⁴ Loc. cit., p. 118.

⁵ Loc. cit., p. 118.

⁶ Corner and Singer, quoting Chavigny, *Ann. de l'Inst. Pasteur*, 1897, p. 680; *La Presse méd.*, Par., 1898, lix, p. 26, and Muscatello and Gangitano, *La Riforma Medica*, Rome, 1898, xiv, pt. 3, p. 471; 1900, pp. 508, 519, 530; *Med. Wochenschr.*, 1900, No. 38; *Centralbl. f. Chir.*, 1900, pp. 112-113.

⁷ One of the most instructive papers is "Morbid Conditions caused by *Bacillus aerogenes capsulatus*," by W. H. Welch, *Bull. Johns Hopkins Hosp.*, 1900, xi, p. 185.

To one who has not a special training in bacteriological work the conclusions are somewhat bewildering. For example, the *Bacillus aerogenes capsulatus* of Welch was at first described as non-motile and anaerobic, but in the later paper by Welch and Flexner it is said to be sometimes motile and not always anaerobic. On the other hand, the *Bacillus œdematis aerobicus* of Sanfelice was motile, but that of Corner and Singer, described as identical with it, was non-motile. Referring to this and other differences between the organisms isolated by Corner and Singer and that found by Dudgeon and Sargent, the last named authors say that such differences are well known to occur amongst the various members of the *Bacillus coli* group, and, as quoted above, they attribute the emphysematous gangrene in both cases to the *Bacillus coli*. But it is certain that there are many conditions attributed to the action of this bacillus which are not associated with gas formation in the tissues, and as to when and why gas is produced in wounded structures by the *Bacillus coli* we seem to have no information. It has been said to occur in diabetic subjects, but it has been observed without this complication.

An explanation of these difficulties is that various species of bacilli closely resembling each other have not yet been differentiated. In this connexion the work of Dr. M. H. Gordon is important. He has recorded that "comparisons between various staphylococci in regard to nine selected actions has shown differences not merely of degree but of kind, and has shown that a differentiation far more elaborate than has yet been supposed to exist naturally obtains amongst staphylococci."¹ Dr. Theobald Smith² has also shown that there are bacilli having many characters in common with the *Bacillus coli*, but different in their reactions in various media, at various temperatures and under other varying circumstances. Such differences might account for the diverse manifestations which have been attributed to the action of one bacillus.

Dr. Eugene F. McCampbell records³ that the *Bacillus aerogenes capsulatus* is very widely diffused, occurring almost universally in the intestinal tract of man and animals, in sewage, soil water, milk, &c. He quotes Dolby, that it was found in 69 of 250 blank cartridge wads. It has been isolated in many pathological conditions, amongst others in

¹ "Report on some Characters by which various Streptococci and Staphylococci may be Differentiated and Identified," by M. H. Gordon, p. 43.

² "Note on *Bacillus coli communis* and Related Forms," by Theobald Smith, M.D., *Amer. Journ. Med. Sci.*, 1895, cx, p. 283.

³ "The Toxic and Antigenic Properties of *Bacterium Welchii*," by Eugene F. McCampbell, M.D., *Journ. of Infect. Dis.*, 1909, pp. 537-563.

cases of surgical operation, and even after such slight injuries as those inflicted by the administration of salt infusions and the giving of hypodermic injections. In one case of bullet wound of the knee it was found in great abundance on the bullet removed from the infected knee by operation. This bacillus has also been found in cases in which the only possible direct communication between the affected parts and the outside of the body was through injured intestine.

The *Bacillus coli* is also very widely diffused. It occurs, of course, in the intestine of animals and man, in sewage water, &c.

It would appear that gas develops much more rapidly from the presence of the *Bacillus aerogenes capsulatus* than from that of the *Bacillus œdematis aerobicus*. McCampbell¹ attributes the toxic, as distinguished from the aerogenic, effects of the *Bacillus aerogenes capsulatus* to a development of organic acids, chiefly butyric acid. No distinct toxins were isolated by the various methods employed, but, in regard to toxicity, butyric acid of the same acidity as the cultures produced the same effect.²

In a fairly extensive examination of the literature of this subject, I have not found any case exactly resembling that now recorded. Dr. James F. W. Ross relates³ the case of a woman, aged 23. Twenty-four hours after her first child was born she was seized with a condition diagnosed as a severe attack of *la grippe*, from which her husband was suffering. She developed all the signs of puerperal septicæmia, with general pelvic inflammation and high temperature. On the twelfth day the uterus was irrigated and packed with iodoform gauze, and on the following day there seemed to be general peritonitis, the temperature rising to 105·2° F., and the pulse-rate to 170. She gradually improved, but later (the exact time is not stated) the abdomen suddenly and rapidly distended. Thirty-four days after delivery the abdomen was opened and found full of gas which had a sweet odour. The intestines were at the back of the abdomen, and there was neither pus nor any collection of serous fluid, nor any sign of peritonitis. The patient died three days later, the abdomen having again become greatly distended.

Except that this case was associated with severe puerperal septicæmia, and that recorded above with a subacute or "cold" abscess

¹ McCampbell, loc. cit., p. 560.

² Loc. cit., p. 562.

³ "Pneumo-peritoneum," by James F. W. Ross, M.D., *Amer. Journ. of Obstet.*, 1895, xxxii, pp. 710-727.

formation, the two closely resemble each other as regards the pneumoperitoneum. In my case the abscess mischief was cured by drainage, in the other the septic uterus remained and death followed. Ross refers to four other cases in which odourless gas collected without fluid and without trauma. All died.

Mr. Godlee (now Sir Rickman Godlee) published three cases¹ of gas and fluid in the peritoneal cavity without perforation of the bowel. In one case colotomy was performed upon a patient, aged 72, for obstruction, which had lasted three weeks. Gas and a little clear fluid were evacuated from the peritoneal cavity, and the patient was alive five weeks later when the case was published. This is the only instance I have noted, except the one now recorded, in which the patient survived an accumulation of gas in the peritoneal cavity without repair of a wound through which the gas had passed into that cavity.

From the large amount and rapid development of the gas in my case, and from the absence of odour, it would seem that the *Bacillus aerogenes capsulatus* was the most likely cause of its presence. That bacillus usually develops in dead tissue, but in this case there was none. The record of Graham, Stewart and Baldwin, and that of Ross above quoted, show, however, that gas does occasionally develop in the living tissues, and it seems probable that the part invaded in the case now recorded was the abscess wall at some point where it was in contact with the peritoneum, and that it possibly consisted of adventitious tissue forming adhesions. But this does not explain why the gas formed in the peritoneal space alone. It seems possible that the gas-producing bacillus was introduced into the peritoneal cavity or into the blood-stream during the operation, and remained alive until the vitality of the abscess wall became so lowered that the bacillus was able to effect in it the characteristic action which, as a rule, it excites only in dead or dying structures. It is fairly certain that the arrest of further development of gases was due to the restored vitality of the lower part of the peritoneal sac brought about by the relief of tension in the abscess and in the peritoneal cavity. As the tissue, the vitality of which was lowered, formed part of the peritoneal sac, gas development in that sac might occur, whilst some unfavourable condition in the abscess cavity prevented its development there. This view would seem to involve the conclusion that the bacillus lived without exercising its aerogenic action for nearly six days. It was shown by Welch and Flexner that the *Bacillus*

¹ "Three Cases of Gas in the Peritoneal Cavity," by R. J. Godlee, *Trans. Clin. Soc. Lond.*, 1877, x, pp. 115-117.

aerogenes capsulatus could live harmless in the circulation for at least forty-eight hours, and, although it was nearly six days before abdominal distension was well marked in the case now recorded, there may well have been a beginning of gas formation much earlier. But this suggestion is purely speculative. In Ross's case gas seems to have developed about thirty days after the injury, that is, the birth of a child, which, however, was followed by various gynæcological manipulations. The outstanding facts now shown are that gases may develop rapidly in the peritoneal cavity, and that this condition may be recovered from.

One cannot consider this case without thinking of the enormous variety of gaseous development within the intestine in individuals, both apart from operations and after operations. It is highly probable that this variation is in part at least due to the presence of aerogenic bacteria. When a sudden great increase of gases takes place within the intestine it would appear likely that some peculiarly active bacillus is present, and as both the gas-producing bacteria that have been described are found in the intestine it would seem to be a not unreasonable conjecture that an unusual development of gas is due to an increased activity of some form of aerogenic organism.

The fact that the *Bacillus aerogenes capsulatus* usually develops in dead or dying tissue, and the invariable association of the *Bacillus œdematis aerobicus* with septic organisms, have acquired a very special interest since this paper was written. The men of our Expeditionary Force in France are constantly fighting in recently made trenches where the opportunities are numerous for infection of wounds by anaerobic organisms which inhabit the soil. Already a number of cases of emphysema complicating wounds have been recorded, and aerogenic bacilli have been isolated from these wounds. It is obvious that to prevent the occurrence of this complication, which is almost always an indication of extreme danger, it is necessary to preserve the vitality of the parts in every possible way, and this necessity adds force to the plea made recently by Sir Watson Cheyne at the Medical Society, and by others, for an intelligent application of the teachings of Lister to the conditions of the present war. Aseptic surgery is useless and impossible when the wounds are made septic as they are inflicted, and if there is a chance that aerogenic bacilli may be present and also the tetanus bacillus, which is another inhabitant of soil, it would seem to be imperative that an attempt should be made to sterilise these infected wounds at the earliest possible moment by a vigorous but judicious use of antiseptics.

DISCUSSION.

Mr. BALLANCE, M.V.O. (Chairman), said that it was a great pity that more members of the Section were not present. Mr. Malcolm's case was most interesting, and the success of treatment very important. It appeared to be allied to, and to be an example in an exaggerated form of, abscess plus gas in the peritoneum, which all surgeons were familiar with and which occurred as the result of disease or of rupture of the intestine. The nature of the organisms present no doubt determined the presence of the large amount of pneumo-peritoneum. Mr. Malcolm was much to be congratulated.

Mr. BATTLE thought, from the history of the case, that there was a possibility that the condition described was due to the introduction of air during the manipulation of the operation. In favour of this was the absence of pus in the air-containing cavity. The abdomen would not be examined for such a complication, and perhaps the onset of localised suppuration near drew attention to the condition of pneumo-peritoneum, which, without this additional complication, would have been overlooked. The escaping air was without odour, and this also favoured this view. Some years ago he published in the *Transactions of the London Medical Society* the notes of a case of pneumo-peritoneum after the removal of a ruptured ovarian cyst. Much heavy fluid had escaped into the general peritoneal cavity, and on the completion of the operation the intestines did not come into the pelvis, but remained compressed against the spinal column and posterior parts of the abdomen. The patient had large peritoneal sacs in both inguinal canals, which had been over-distended by the fluid. Air could be demonstrated in these for some three weeks after operation and only gradually became absorbed. The girl had cardiac disease, and it was not considered well to prolong the operation. In his experience these cases of cylindroma of rectum gave very different results. Quite recently a man, who came to the hospital for heart disease, presented a card which showed that Kraske's operation, performed ten years ago for extensive growth, had been successful in restoring perfect function and there had been no recurrent growth. In another, where the disease had been very considerable, the patient (a young man, aged 19) was still hopeful after more than three years. In a third, where the growth had been far less extensive, the man died about nine months later from secondary growth in the anterior part of the brain, whilst the pelvic organs appeared quite healthy.

Mr. MALCOLM, in reply to Mr. Ballance, pointed out that in the case recorded the gas-containing cavity and the pus-containing cavity were quite separate, and there was no evidence of inflammation in that part of the peritoneal sac in which the gas collected. The patient's highest temperature during the five days before the gas and pus were let out was 99'4° F., so that the abscess was described as "cold." His own experience did not include many cases in which gas and pus were found together in the peritoneal cavity, and he did

not remember any in which the gas did not obviously come from outside or from the bowel. It appeared that the gas-producing power of micro-organisms was greater when the vitality of the tissues was low, and it was said that 80 per cent. of the cases arose in wounds of the extremities. Everything, therefore, must be done to preserve vitality, to reduce tension in wounded parts, and to prevent septic organisms from gaining a hold. Mr. Battle's case of air in the peritoneal sac was very interesting, but in the case now recorded the gas certainly increased in quantity, whilst there was no possible channel by which air could enter, as the wound in the bowel was sealed off by suppurative inflammation. Mr. Battle's case reminded the speaker of one in which after removal of a very large ovarian tumour the small intestines were found bound down by adhesions in a small space at the back of the peritoneal cavity. At that time, many years ago, air was considered a source of sepsis, and, therefore, every effort was made to squeeze as much as possible out before closing the abdomen. The patient died, and the peritoneal sac was found filled by an enormously distended large intestine. Clearly it would have been wiser to have left some air in the peritoneal sac.

The Black (Pigmented) Appendix.

By WILLIAM HENRY BATTLE, F.R.C.S.

IN the *Lancet*¹ for 1913 I described a series of four cases in which a very unusual amount of pigmentation of the mucous membrane of the appendix was found after its removal for disease. Three of them were sketched, and I am able to produce the water-colour sketches for the inspection of the Section; the fourth appendix was mounted by Mr. Shattock, and is also shown. It differs from the others in the fact that there has been a great destruction of tissue towards the distal extremity. With the exception of the case of extensive pigmentation of the large bowel, due to *lead* deposit, described by Dr. G. Newton Pitt,² I did not know of any similar appearance at that time. Recently, however, the subject of pigmentation of the bowel has been referred to by Sir J. Bland-Sutton,³ who found a very blackened state of the mucous membrane of the bowel in a woman suffering from obstruction due to cancerous growth of the large intestine. The cause of this pigmentation could not be ascertained, for the patient recovered. He refers in that

¹ Battle, "The Black (Pigmented) Appendix," *Lancet*, 1913, ii, p. 135.

² *Trans. Path. Soc. Lond.*, 1891, xlii, p. 109.

³ Presidential Address at the Medical Society of London, *Lancet*, 1914, ii, p. 934.

address to an article by Dr. Ludwig Pick,¹ which had escaped my notice, in which Dr. Pick refers to two cases of pigmentation found in English publications (besides that of Dr. Pitt), both of which were caused by the deposit of *mercury* in the tissues. (1) The case of a woman, aged 74,² who had taken mercury regularly for forty-three years. Here the submucous tissue of the large intestine from the ileo-caecal valve to the rectum was mottled like the skin on a toad's back. The patient had rectal prolapse before death, and the mucous membrane was seen to be deeply pigmented when extruded. (2) The case of a man, aged 64,³ who had been under treatment for syphilis. He exhibited a similar pigmentation of the large bowel dependent on a deposit of mercury in the mucous membrane. In this case, which was described by Dr. Rolleston, there was no evidence of pigmentation during life.

The contribution to the subject which I now wish to make consists of two cases of marked pigmentation of the appendix met with in women of different ages and circumstances. Their residences were far apart, in Berkshire and Worcestershire, whilst of the four other instances already published, three came from within London and one from Kent. The cause of the phenomena must therefore be very widely distributed.

The first case is that of an unmarried lady, aged 48, who was seen in consultation with Dr. Guilding on April 30, 1914. The medical history was a long one. Since the age of 13 she had been delicate, and for years past constantly in the hands of the doctor. Since the age of 21 she had always suffered from constipation, and the bowels had not acted without the use of purgatives. She had complained of indigestion for years, sometimes being worse and sometimes better; of late years she had of necessity been very careful as regards her diet. In 1899 the cervix uteri was divided for dysmenorrhœa. In 1900 she had symptoms of inflammation of subacute character, which were said to have had their origin in the right ovary and broad ligament. Ever since the operation in 1899 she had had occasional attacks of pain and tenderness in the right iliac fossa, during which there had sometimes been vomiting. In some of these attacks the pain was more in the upper part of the abdomen. In 1904 she had an attack which was

¹ *Verhandl. d. Berl. Med. Gesellsch.*, 1911, p. 161.

² C. J. Williams, *Trans. Path. Soc. Lond.*, 1867, xviii, p. 111.

³ H. D. Rolleston, *Trans. Path. Soc. Lond.*, 1892, xliii, p. 69.

described as "acute ovaritis," on the right side, and had felt "uncomfortable" on that side since. In 1911 she had an attack of "neuritis" in the right arm, which lasted nearly the whole year. She was also under treatment for colitis and a toxic condition towards the end of that year, and this was resumed for varying periods during 1912 and 1913, finishing in March, 1914. Before 1914 the right iliac fossa was not much in evidence; after the beginning of that year she had four attacks of pain and tenderness in that region, one of them being severe and accompanied with a rise of temperature and other signs of inflammation, but without vomiting. She had become thin, sallow, and pale, with a tenderness persisting over the appendix region. The diagnosis previously given was that of a chronic appendicitis with probable ileal kink and chronic intestinal stasis.

An examination by the X-rays after a bismuth meal had been carried out, and the following points were established:—

(1) A delay in the passage of the bismuth through the cæcum.

(2) The ascending colon and transverse colon were distinctly dropped, and there was considerable stasis in the passage of the bismuth all along the large bowel.

(3) Considerable loss of tone in the rectum. Here the bismuth lodged and distended the part, having to be evacuated by means of enemata.

When I saw her in April I agreed with the diagnosis of subacute inflammation of the appendix, with want of muscular power in the large bowel and rectum. She was rather feeble, delicate, and "tired-looking." The abdomen was slightly distended, generally tympanitic, and tenderness was complained of on examination of the right iliac fossa.

Operation was performed on May 2, 1914. The appendix was about $3\frac{1}{2}$ in. long, slightly adherent at the top. The meso-appendix was shortened at one point, and there were chronic catarrhal changes with submucous fibrosis. The mucous membrane was of a uniform brownish-black colour due to the deposit of pigment. From the abrupt termination of this at the place where the appendix had been cut off it was quite evident that a similar condition extended into the cæcum. The intestine did not give any evidence of this on inspection. The right ovary was atrophied and the tube normal. A membrane united the transverse and ascending portion of the colon at the hepatic flexure, making a somewhat sharp and fixed bend. It appears to have been derived from the right margin of the omentum. This membrane was

divided and a few vessels in it ligatured. The cæcum was somewhat larger than usual, with a tendency to drop towards the pelvis. The ovary and tube were removed.

Recovery was uneventful.

The second case was also met with last year. On May 17, 1914, I operated for Dr. Brockatt, of Malvern, on a young unmarried lady, aged 27, who gave the following history: Five months before she had had an attack of appendicitis, which lasted five days. Two months before she had suffered from a second attack, which lasted seven days. There had been some occasional pain between the attacks but otherwise she had not been ill. She always suffered from constipation. Nothing abnormal was felt on examination, but there was some tenderness in the right iliac fossa, and she stated that pain had been present there the previous evening. The temperature was normal. The appendix, when removed, was found to be $5\frac{1}{2}$ in. in length, bulbous, and red at the top from recent inflammation. The meso-appendix was shortened at one point. The mucous membrane was pigmented, the shade deepening in intensity to the line of section where it was quite black. There was submucous fibrosis. No adhesions were present. No other abnormal condition was found. This appendix was lost.

These two cases, together with those already published, make a series of six which present some common features. In the first place, there was an extensive brownish-black pigmentation evidently not limited to the appendix, but spreading an unknown distance into the large bowel. The patients were all of the female sex (aged 27, 30, 32, 43, 48, 48) and in each there was a history of constipation of long standing, which amounted to intestinal stasis in more than one of them, according to their medical attendants. It would appear as if in some the difficulty with the bowels had led to a secondary inflammatory affection of the appendix. Operation in each was for the relief of pain caused by this. The pigmentation was found on examination of the removed appendix, and in only one was there any appearance of darkening of the bowel—as examined from the operation incision. Microscopical examination showed the deposit to be both in and between the cells of the mucous membrane (Shattock).

Chemical analysis of the first three specimens by Dr. Le Sueur showed that the pigmentation was produced by a deposit of iron, the exact combination not being defined owing to the small quantity of

material available. There was no bismuth, lead, or mercury present. In only one patient was there any history of the taking of iron medicine—and she had been given the compound syrup of the hypophosphites ($\frac{3}{4}$ grm. ferri hypophosph.).

There is very little, if any, tendency to pigmentation of intestine by iron given in the form of medicine, otherwise the condition would be frequently met with in the post-mortem room, and we should have had many references to it in our medical publications. From the fact that I have met with these six cases within a comparatively short period, there is a probability that it is more common and widely spread than we have suspected. The question arises as to the source from which the iron has been obtained. There can, I think, be very little doubt that it is derived from wheat flour prepared by the iron rollers which are used in the modern process of milling. These rollers are fluted, and the fluting wears down somewhat quickly, and although measures are taken to prevent iron particles from entering the mill none are taken to remove the iron dust and small particles which come away as a result of the rubbing of one roller against the other. The method of grinding with iron rollers was once applied to the manufacture of light-coloured pigments for painting walls, but had to be given up because of the brown patches which appeared on the walls to which such paint was applied. The rollers wear away more quickly in the grinding of wheat, especially that grown abroad, which is harder than ours, than in the preparation of pigment. Chemical examination of a quantity of best flour has shown the presence of an abnormal amount of iron, although none could be separated from the flour by means of a magnet.

I have already alluded to the fact that no measures are taken to extract the iron from flour prepared by these rollers, therefore there is a considerable quantity being taken into the alimentary canal by the population at large. It is very probable that the increased amount of troublesome constipation of recent years, which in some cases is described as "stasis" and ascribed to a kink of the bowel, is due to this cause. Kinks possessing a clinical importance are in my experience very rare.

It would be interesting to know from the experience of the physicians whether anæmia is any less common than it used to be.

I think that it may be possible that the formation of fæcal concretions is assisted by this ingredient of flour, but it is not possible to say much on the subject. I have submitted several concretions to the chemist for examination but the result of examination for iron is negative—they appear to contain very little.

The question of the influence of roller-milling in the causation of appendicitis is one which has interested me for some years. Facts are accumulating to show that it is a serious factor, but this is not the occasion on which to consider it. There is, however, very little doubt that in addition to the small sharp particles which result from the disintegration of the fluting of the rollers, and which sometimes cause localised ulceration of the mucous membrane, there is a considerable amount of "iron dust" which is given out in such quantities that in certain individuals it can produce an actual blackening of the mucous membrane over extensive portions of the large bowel. This pigmentation is more extensive than could be produced by blood extravasations, and is more uniformly diffused.

DISCUSSION.

Mr. BALLANCE, M.V.O. (Chairman), said that Mr. Battle's paper raised many points of much interest, both from the surgical and public point of view. It was difficult to see why the appendix should be selected for pigmentation. Pigmentation was usually caused by some pigment derived from the blood, and it did not seem certain that in these cases gross particles of steel were lodged in the tissues of the appendix. Mr. Battle's further researches would, no doubt, clear up many points which were still obscure.

Mr. MALCOLM had seen a number of pigmented appendices. He had thought that this was caused by some deposit of blood pigment, but, frankly, he did not understand what the colour was due to, and he felt much indebted to Mr. Battle for an explanation. He had seen cases in which a pigmentation seemed to be localised in parts of the mucous membrane of the appendix, which was not easily explained by Mr. Battle's view, but was also difficult to understand by any other. The condition had become more interesting since this interpretation had been given.

Mr. BATTLE, in reply, said that he had been much interested by the remarks of Mr. Ballance and other members of the Section. He did not think that the cases referred to by some could have been of the same nature as those which he had shown. The pigmentation in his cases was so deep that special attention must have been drawn to it. Personally, he had only met with these cases out of very many examined. As regards Mr. Ballance's question as to the possibility of such a state in the appendix of a Russian on whom he was going to operate, he was afraid that the data were insufficient. He was inclined to attribute the origin of the iron to the preparation of the flour by roller-milling, whilst its influence in the causation of appendicitis was shown by the experiences of a provincial town which had only presented cases of that disease since the local millers had substituted iron rollers for the granite setts formerly in use.

A Case of Obliterative Arteritis.

By CHARTERS J. SYMONDS, M.S.

MR. B. came under my care, at the age of 35, for commencing gangrene in the left great toe, and gave the following history: In 1907, at the age of 29, he injured his left leg in a fall from his horse; he had another injury a few days later, when he twisted his right ankle and was laid up two or three weeks. Before the graze on his left leg had healed he developed phlebitis above and below the injury; after complete recovery the phlebitis recurred, and gave so much trouble that he was advised to return home from India where these accidents had occurred. He arrived in January, 1908, and within six months he was laid up three or four times with phlebitis in one leg or the other both above and below the knee. In the summer of 1908 portions of the internal saphena veins were removed just below the opening. His blood at this time was also examined without any information being obtained. In the autumn and winter of 1908 he spent a good deal of time in the Spithead Forts, where he suffered from cold, especially in the feet. As the weather got warmer his feet, which had troubled him a great deal, gradually improved. In July, 1909, the right great toe became painful, and what he calls a "crack" appeared on the end below the nail. He continued his work for a couple of weeks and was then obliged to lay up in the month of September. From this time he has never returned to military duties, and has practically been an invalid. The condition of the toe got worse, suppuration ensued, and in August, 1909, some diseased bone was removed, and the aperture plugged daily for nearly three months. He had a great deal of pain at this time in the toe, which was relieved after removal of the nail. As, however, the wound did not heal, notwithstanding the most careful treatment, the great toe with the metatarsal bone was removed in the winter of 1909-10. The wound healed and in March he went to Algiers. The following winter he spent in Egypt, and 1911-12 in Mentone. The fingers began to give trouble in 1909; the ulceration appears to have started from a slight injury while cleaning a bicycle. Part of one of the nails was removed, and Bier's cup applied to the index-finger of the right hand. Then the index-finger of the left hand became ulcerated at the tip while he was in Egypt in 1911; it healed quickly by dry heat in

an electric box. The fingers have continued to give trouble both in 1913 and 1914.

In September, 1912, Mr. Clowes, in writing and asking me to see this patient, said that three weeks before he wrote a blister appeared on the dorsal aspect of the left great toe, attended with a great deal of pain.

When I first saw him the left great toe was swollen and of a dusky red colour. Thin discharges escaped from under the nail. The pulp of the toe was black, and it was evident that gangrene was beginning. The pain was so great as to demand opiates, and was felt in the whole foot and up the leg. He was so broken in his nervous system that any examination was difficult. One could make out, however, that the pulsation in the femoral was poor, nor was it good on the opposite side. The other toes were tender and the circulation in them was feeble. Removal of the nail gave some relief, but the gangrenous process continued to spread, remaining, however, confined to the great toe. Yielding to his appeal, the toe, with the metatarsal bone, was removed. There was practically no bleeding, and it was obvious that a higher amputation must be made, more especially as in the next few days the other toes showed signs of failing circulation and the dorsum of the foot became dusky. The question as to the site of amputation was an important one. The femoral pulsation was fairly vigorous, but the wall of the vessel appeared thickened. No pulsation could be felt in the popliteal or tibial arteries on either side. As the process was slow I decided to amputate through the middle of the leg by lateral flaps. If on finding the main vessels occluded the anastomotic evidence was good, to be content; but if no bleeding took place then to remove the limb above the knee. Sir Alfred Pearce Gould, who saw the patient with me at this time, thought it wiser to amputate above the knee at once. He, however, concurred in the course I proposed to take. On cutting through the soft parts, the posterior tibial was found to be completely occluded, the anterior yielded some blood, the smaller vessel bled fairly well. The amount of vascularity seemed to justify the site selected. Healing followed fairly well, practically a primary union resulting. The edges of the flaps remained a little dusky and tender for a time, but ultimately he obtained a well-nourished, useful stump, the condition of which to-day is quite satisfactory.

The right foot from time to time gives trouble, and he has phlebitis occasionally, so that he is unable to take much exercise. The fingers at the present time are tender though healed. The second toe of the right foot has been tender and even "inflamed" since August, 1914, and the third toe has more recently been tender and red.

Examination of the vessels: The posterior tibial artery was a hard cord, the lumen completely occluded. It was free from adhesion to surrounding structures, as was the anterior tibial. On section it showed the appearances typical of obliterative arteritis, and in an advanced stage. The change begins in the subendothelial layer of the intima, and, progressing inward, occupies the entire lumen. In places the elastic lamina has disappeared, and the muscular layer is invaded by a new-celled growth. The vessels in the granulation tissue occupying the lumen are large and run with the axis of the artery. In the lower part of the artery the wall is almost destroyed and is blending with the surrounding tissues. The wall of the vein is also thickened.

CAUSATION AND PATHOLOGY.

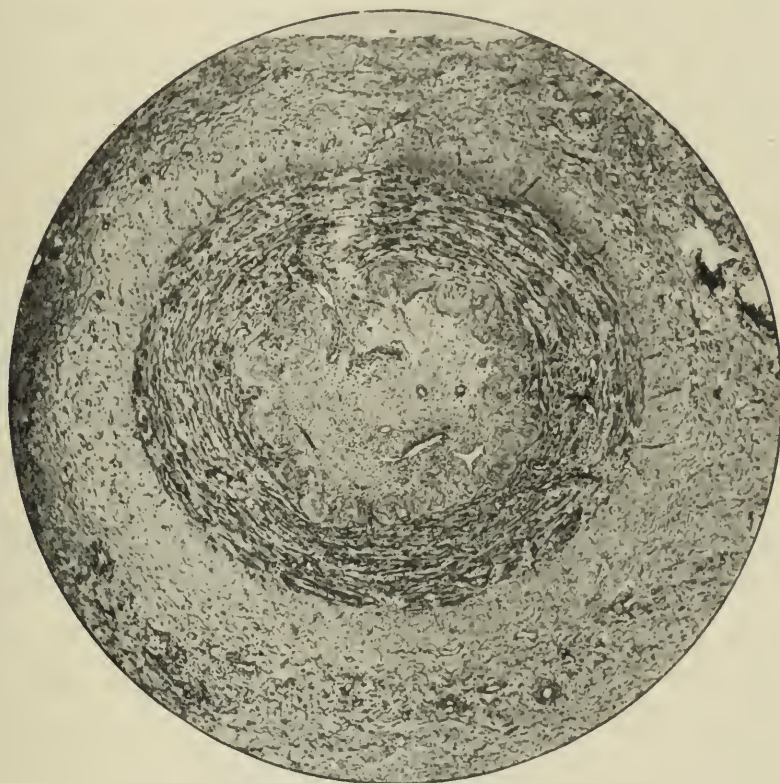
A syphilitic basis for the disease in this case has not been discovered. A Wassermann reaction while the disease was in progress, made by Dr. Eyre in December, 1913, was negative. Dr. Eyre says not even a faint positive reaction due to previous malarial disease which occasionally confuses the issue was observable, but the opsonic index to the streptococcus was low. Previous to this he had had a long course of biniodide of mercury without any appreciable benefit. Arsenic has been tried without any marked result, so that one must leave the causation undetermined. That the disease is still progressive is shown by the condition so late as October, 1914, when the tips of four fingers were tender and at times showed superficial ulceration. Most of these followed slight injuries, such as a prick of a pin; the use of Bier's cup generally affords relief. The condition of the second toe on the right foot also indicates further changes in the vessels of this limb, and in writing he says that appearances were very much like those which preceded the trouble in the left foot two years ago, so that the outlook is by no means good.

What connexion the phlebitis, which he has had off and on for some years, and which preceded the graver disease, has with the arterial condition it is impossible to say. The walls of the veins as seen in the sections are in places distinctly thickened. He gives a history in his earlier years of "dead fingers" and chilblains on his toes, and he thinks on his fingers; there are no other signs of Raynaud's disease, nor is there in his family any condition allied to that from which he suffers.

In the Clinical Society's *Transactions*,¹ vol. xvii, is a case recorded

¹ *Trans. Clin. Soc. Lond.*, 1884, xvii, p. 95.

by Sir A. Pearce Gould, on February 8, 1884, of a spreading obliterative arteritis of the right brachial artery in a male patient, aged 19. There was dry gangrene of the tip of the thumb and of the fourth finger, and the boy suffered a great deal of pain. The progress of the disease in the artery could be traced as it gradually spread upwards. In this case the radial artery had shrunk so as to be only just perceptible, and the brachial artery was pulseless up to 1 in. below the lower border of the *teres major* muscle, and above that point the vessel was harder than



Transverse section of the posterior tibial artery.

the opposite side. His patient recovered the use of his arm and was able to return to his work.

In a note in vol. xx of the same Society's *Transactions*, made in October, 1886, some further extension of the disease was noted. The patient had remained at work till six months previously, when he was attacked with numbness in the right arm and forearm. Later he lost power in the left arm and became unconscious for a short time. In Sir Alfred's remarks will be found references to other cases.

My object in recording this case is to bring before the Section an example of what appears to be an extremely rare condition of the

arteries for which no sufficient explanation has been found. Though the disease is still in progress it seemed worth while to bring the case before the notice of the Section. As to the special part of the vessel affected it is interesting to compare the changes that take place in syphilis and in arteries after ligation and also in suppurative arteritis.

The specimen under the microscope shows the changes very well, and may be compared with that of an artery thirty-two weeks after ligation and also with specimens of suppurative arteritis which were published in the *Transactions of the Pathological Society of London*, 1884, vol. xxxv, p. 146, and here shown with the epidiascope.

Postscript.—I had an opportunity of examining the patient on February 6, 1915. The stump was in excellent condition, well nourished, of good colour, and free from tenderness. He was able to wear the artificial limb for some hours each day. The second toe of the right foot was blue, enlarged, and was often tender. From time to time he says it becomes painful and still further enlarged. It is the liability to this swelling which limits his walking. The other toes are dusky. Neither the posterior nor anterior tibials can be felt. The femorals pulsate well on both sides. The fingers of both hands were blue from the last two joints. The morning was cold, and he had been sitting still, reading. On one finger there was suppuration round the nail. This had been preceded by one of the small, sensitive ulcers on the end of the finger, and appeared to me due to local infection and not to its vascular condition. A similar onychia had existed on the adjoining finger, the effects being shown in the condition of the nail. He uses a Bier's cup with benefit. The radial arteries pulsate fully; the vessel wall, however, is hard and appears thickened. Neither ulnar can be felt. The heart does not appear enlarged, there is no abnormal sound, and the apex beats well within the nipple.

Mr. BALLANCE, M.V.O. (Chairman), said that the origin of the obliterative arteritis in Mr. Symonds's case was not known. The case reminded him of that recorded by Sir A. Pearce Gould and the case recorded by the late Dr. Hadden. The arteritis obliterans of Friedländer and von Winiwarter was of the same type. In all these cases the artery was slowly obliterated by the same process, namely, by a thickening of the intima. It naturally occurred to him to ask whether the onset of gangrene might be diverted by reversing the circulation in the limbs—by arteriovenous anastomosis.

PROCEEDINGS
OF THE
ROYAL SOCIETY OF MEDICINE

VOLUME THE EIGHTH

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1914-15

PHARMACEUTICAL & PHARMACOLOGICAL SECTION



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LONDON
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1915

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THERAPEUTICAL AND PHARMACOLOGICAL SECTION.

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The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

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Therapeutical and Pharmacological Section.

January 19, 1915.

Dr. HALE WHITE, President of the Section, in the Chair.

Action of Suprarenals on Renal Secretion.

By DOUGLAS COW, M.D.¹

IN a previous paper² I showed the presence of a direct vascular connexion between the medullary portion of the suprarenal bodies and the kidneys, by which path adrenalin could (and under certain conditions actually did) pass directly from the suprarenal bodies to the kidneys, without previous dilution or oxidation in the general circulation. As a result of observations on the effect of suprarenal stimulation on the flow of urine, I came to the conclusion that the suprarenal bodies acted as direct regulators of renal activity, without, however, having studied the effects on the kidney in detail. The observations on which this conclusion was based were that: (1) Stimulation of the suprarenal body produced an abrupt shutting off of the flow of urine on the same side; (2) a momentary increase in the rate of flow of urine took place before this abrupt cessation; (3) the diminution in the flow of urine was out of all proportion to any change in the amount of fluid perfusing through the kidney vessels.

I have since studied the effects of suprarenal stimulation on the kidney in greater detail. A series of cats were anæsthetised first with A.C.E. mixture, and then with urethane (1 gr. per kilo. of body-weight). Special precautions were taken in order that the animals should not be frightened before being rendered unconscious.³ The animals were

¹ Beit Memorial Research Fellow. From the Pharmacological Laboratory, Cambridge.

² Cow, *Journ. of Physiol.*, 1914, xlviii, pp. 5, 443.

³ Elliott (*Journ. of Physiol.*, 1912, xlv, p. 374) has shown that fright or stimulation of any sensory nerve produces a pouring out of the adrenalin content, with consequent exhaustion of the suprarenal bodies.

2 Cow : *Action of Suprarenals on Renal Secretion*

then eviscerated (stomach, intestines, and spleen); the suprarenal on one side, usually the left, was removed after being completely ligatured off; and the sciatic nerve on one side was exposed and placed on electrodes.

The main suprarenal vein on the side on which the suprarenal body was left intact was then clamped at its point of entry into the inferior vena cava, in order to prevent the products of activity of this gland from finding their way directly into the general circulation; and the sciatic nerve was stimulated intermittently for ten to fifteen minutes. The kidneys were then removed, cut open longitudinally, and at once plunged into fixative. Various methods of fixation were employed:—

- (1) Instant plunging into hot Zenker's fluid at 90° C.¹
- (2) Instant plunging into hot water at 90° C.¹
- (3) Instant freezing, with subsequent thawing in a fixative solution.

Whatever method of fixation was employed, the two kidneys of any one animal were always treated in exactly the same manner both as regards fixation and subsequent processes.

In all cases it was found that the kidneys through which the products of activity of the suprarenal bodies had passed presented the following characteristics, as compared with their fellows, which had not been so acted upon:—

- (1) The kidney, as a whole, was distinctly shrunken.
- (2) The glomeruli showed a peculiar condition, the capsule of Bowman being contracted tightly over the glomerular tuft.
- (3) The tubules were constricted.
- (4) The intertubular tissue appeared to be stretched, spaces tending to appear between individual tubules.

Direct measurements of the kidneys were taken both before and after sciatic stimulation by means of calipers. The following table gives these figures from one of these experiments:—

LEFT KIDNEY (left suprarenal removed).				RIGHT KIDNEY (suprarenal intact).			
		Before stimulation	After stimulation			Before stimulation	After stimulation
Length	...	37 mm.	35 mm.	Length	...	40 mm.	37 mm.
Breadth	...	20 "	22 "	Breadth	...	20 "	19 "
Depth	...	20 "	18 "	Depth	...	22 "	18 "
Cubic	...	14,800	13,860	Cubic	...	17,600	12,654
<i>Reduction</i>	...	6 per cent.		<i>Reduction</i>	...	28 per cent.	

¹ Recommended by McGill for those cases in which it is desired to fix a tissue with as little interference as possible with the condition of the tissue.

Whilst I fully realise the roughness and possible inaccuracy of this method of gross measurement, the results were in agreement in the various experiments, and also with the corresponding microscopic findings; and, failing a complicated and practically impossible oncometric experiment (it would be necessary to include in an oncometer not only the kidneys and their vessels, but also the suprarenal bodies and their blood supply and nerve supply, which are both fragile and diffuse), it is the only available method.

Various slides and specimens were shown illustrating these results.

SUMMARY.

The products of suprarenal activity which under certain conditions find their way directly to the kidneys produce.--

- (1) Shrinkage in size of the kidney
- (2) Abrupt diminution of urinary flow.
- (3) Contraction of the capsule of Bowman on the glomerular tufts.
- (4) Constriction of the uriniferous tubules.

Therapeutical and Pharmacological Section.

March 16, 1915.

Dr. HALE WHITE, President of the Section, in the Chair.

A Note on the Antagonism of Drugs.

By A. R. CUSHNY, M.D., F.R.S.

THE antagonism of poisons has been a subject of speculation since the earliest dawn of history, and within the last half-century has received a good deal of attention from experimental observers. If one includes the antagonism of toxins and antitoxins, it might almost be said to have formed the chief subject of medical investigation for some years at the close of the last century and the beginning of this one. But while very exact investigations of the quantitative relations between toxins and antitoxins have been made, there are very few similar observations to be found in regard to the antagonism of the ordinary alkaloids. It has been my lot to furnish some of these in regard to the atropine group and pilocarpine, as I had to employ the method of antagonism in investigating the power of optical isomers at different times, and at the close of a further research of this character, I have decided to put together some points which seem to have some interest apart from the special question which I was working on.

My experiments have been performed on the salivary secretion in dogs in which a permanent salivary fistula had been established some weeks or months before, by bringing a submaxillary duct to the surface and suturing it to the skin. The wound had completely healed in each case before the experiments began, and the dogs were all healthy. Each series of observations was made at the same hour and at a regular

interval after the animal had been fed and exercised. The animal lay quietly on its side and the saliva secreted through the fistula in each five minutes was collected on a pledget of cotton-wool; this was weighed before and after, and the difference gave the amount of saliva. After the normal rate of flow had been ascertained several times, atropine was injected hypodermically in the flank, and after its effects had been noted, pilocarpine was injected in the same way and the measurements were continued for forty to sixty minutes or longer; the interval between the injection of atropine and pilocarpine was the same throughout a series, but differed in the different series.

My records extend over four dogs, which have been carefully examined, and I find that in each of these 0.5 mg. atropine sufficed to neutralise the effects of 5 mg. pilocarpine, so that it may be taken that the ratio of atropine to pilocarpine is constant in a series of different individuals.

The next question was whether this ratio is maintained when in the same animal the actual amounts of the alkaloids vary. If x neutralises y , will nx neutralise ny ? This was answered by a series of experiments, which is shown in the following table:—

Atropine in milligrammes		Pilocarpine in milligrammes		Saliva in 20 mins.		Saliva in 40 mins.
0.1	...	1	...	0.19 gramme	...	0.58 gramme
0.2	...	2	...	0.21	„	0.45
1.0	...	10	...	0.00	„	0.34
2.0	...	20	...	0.02	„	0.18
0.0	...	1	...	6.47	„	9.94

Here the antagonism is fairly equal throughout, although the actual amount of the alkaloids is increased twenty times, because the alkaloids are present in the same ratio throughout—namely, one of atropine to ten of pilocarpine.

In another set of experiments the antagonistic action of atropine and pilocarpine was the same whether atropine was given in 0.1 mg. or in 5 mg., provided the pilocarpine changed simultaneously from 1 mg. to 50 mg. In other words, the antagonism of the alkaloids remains unchanged, provided the relative quantity administered is the same, however much the actual amounts may alter. If x neutralises y , then nx neutralises ny .

This sounds strangely like the law of multiple proportion of chemistry, and results similar to those I have obtained for atropine and pilocarpine have, as a matter of fact, been used to show that toxins and

antitoxins form a chemical union and thus neutralise each other. Now there can be no question that the antagonism of the two alkaloids is not due to the formation of an inert chemical compound between them. The confusion arises from the use of the word "neutralise," which must mean absolute neutrality if the chemical law is to hold. This absolute neutrality we cannot gauge in regard to either drugs or toxins. In titrating an acid with a base, if we find that x acid neutralises y base exactly, we know that nx acid will neutralise ny base because we have a very definite endpoint. If, however, we take an approximate endpoint, for example if the true endpoint is x , but we measure off $x + a$, in which a is a trifling error, then when we come to add $n(x + a)$ to ny , we may find the result far from neutral, because na is a considerable amount, and na has the same effect as if neither nx nor ny were present. In my experiments no such definite endpoint was attainable, but the difference from the law of multiples of chemistry is shown by a series of observations.

		Atropine in milligrammes	Pilocarpine in milligrammes		Saliva in 20 mins.		Saliva in 40 mins.	
Experiment 1	...	0.1	...	1	...	0.19 gramme	...	0.58 gramme
„ 2	...	1.0	...	10	...	0.21 „	...	0.45 „
„ 3	...	0.1	...	2	...	3.61 „	...	5.17 „
„ 4	...	1.0	...	20	...	0.37 „	...	3.11 „
„ 5	...	0.0	...	1	...	6.47 „	...	9.94 „

Here let us assume for the moment that in experiments 1 and 2 the antagonism is complete, and that the pilocarpine and atropine neutralise each other in the same way as an acid and an alkali; there was therefore an excess of 1 mg. of pilocarpine in experiment 3 and of 10 mg. in experiment 4. Yet the secretion in experiment 3 is not comparable to that observed from 1 mg. of pilocarpine given alone, and that in experiment 4 (from 10 uncombined milligrammes) is even less than that in experiment 3. In other words, the excess of pilocarpine which is present in experiment 4 does not induce the effects which it would do if given entirely alone, because its activity is modified by the presence and activity of the atropine. The atropine thus continues to exert an influence on further quantities of pilocarpine even though there is enough pilocarpine already to neutralise its action. In other words, the antagonism is an example of mass action and not of the chemical law of multiples.

As regards toxins and antitoxins, the evidence is conflicting; in some cases there seems to be reason to accept an actual chemical

combination, in others this is far from proved, and some facts speak in the contrary sense. The view that toxins form a chemical combination with antitoxin is not based exclusively upon observations of this kind, and I would not be considered to be in opposition to this very widely held opinion. But one of the arguments on which it has been based appears to need further confirmation.

Therapeutical and Pharmacological Section.

April 20, 1915.

Dr. F. RANSOM in the Chair.

Discussion on the Treatment of Phthisis by the Induction of Pneumothorax.

Opened by THOMAS D. LISTER, M.D.

MY chief object this afternoon is to demonstrate an apparatus designed more especially for use in private practice or in the remote country sanatorium. I have been asked by the Secretary to suggest, incidentally, some of the points upon which a consensus of opinion is desirable. No doubt others will occur to those taking part in the discussion.

I hope to bring before the Section certain practical problems arising in the treatment of cases of pulmonary phthisis by artificial pneumothorax, and my own attempts at a partial solution of them. Personally, I shall be satisfied if our discussion this afternoon affords me grounds for altering my methods. On the other hand, I shall be pleased if some of the suggestions I make are favourably received.

I need not enter into a relation of the early history of the operation for artificial pneumothorax. For those who are anxious to study this matter, the writings of Rist in the *Quarterly Journal of Medicine* of January, 1913, and the various abstracts of the history that have been incorporated in many subsequent papers, notably by Dr. Lillingstone, Dr. Vere Pearson, Dr. Clive Riviere, Professor Saugman, and others, will afford sufficient material. At this date it is rather a question of correcting errors arising from individual experience than of deciding as to the utility of the method. Anyone who has seen the dramatic cessation of clinical symptoms in the case of a successful pneumothorax will have no doubt as to the latter.

I propose briefly to demonstrate the apparatus here exhibited with a few remarks as to what I consider its advantages, and to put before the Section for discussion certain questions as to technique which, no doubt, those who have great experience in this method will be able to elucidate and supplement.

THE GENERAL NATURE OF APPARATUS FOR THE OPERATION.

The apparatus consists essentially of (1) a reservoir or reservoirs for the gas or gases used, which are oxygen and nitrogen; (2) a means of displacing the gas from these reservoirs by means of running water or an antiseptic solution into them; (3) a means of conveying the gas into the chest by a tube leading from the gas bottle to a hollow needle; (4) a manometer in connexion with the latter; (5) a means of disconnecting the manometer from everything but the needle in the chest, so that the pressure attained at any moment can be observed when there is no hydrostatic pressure on the manometer from the gas bottles and fluid reservoir, and no interference with the rhythmical variations of pressure due to respiration.

THE APPARATUS EXHIBITED.

The apparatus exhibited is based on that suggested by Dr. de Carle Woodcock, whose method of preparing the gas in the apparatus itself is adopted. His apparatus was made by Messrs. Reynolds and Branson, of Leeds, whom I therefore got to make mine. It differs from his in that it prepares oxygen as well as nitrogen, and is fitted with a regulating tap which controls both the flow of water from the reservoir to either gas bottle and the flow of gas from either gas bottle to the delivery tube, by a single movement. This regulating device is fitted with an indicator showing by a pointer what is being done at any moment. Although cased for convenience in carrying, the whole apparatus is placed on an open stand, and the manometer is brought forward on the table wherever convenient when the apparatus is placed beside the patient. This open form of apparatus is, I consider, more practical in use, and more in accordance with laboratory methods. Artificial pneumothorax essentially demands a laboratory sense as a method of treating disease. In addition to the apparatus for preparing nitrogen based on that of Dr. de Carle Woodcock, but with a new form of bottle for removing oxygen from the air, I have also attached a means of filling the oxygen bottle by preparing it on the spot by the use

of an effervescent mixture. I have to acknowledge the great care and courtesy of the firm in carrying out suggestions, and in making certain experiments beyond my facilities, notably in glass-blowing.

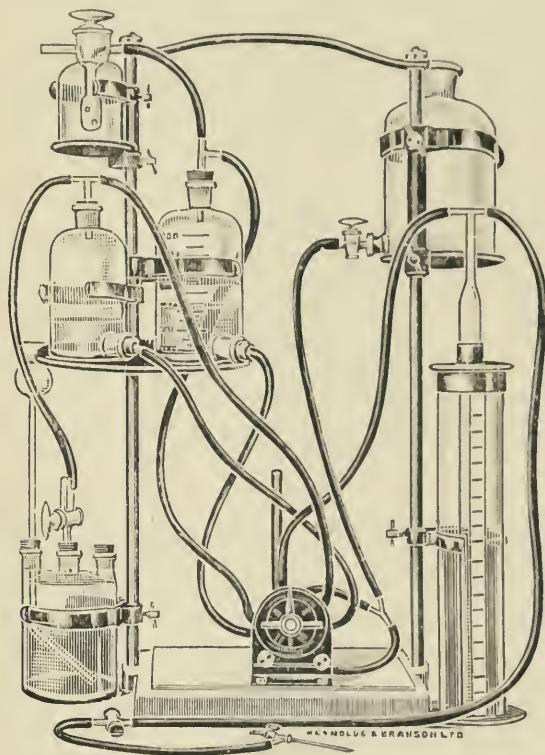


FIG. 1.

A general view of the apparatus demonstrated. A thin gas-bag is interpolated between the Wulff's bottle and the oxygen bottle in the latest pattern.

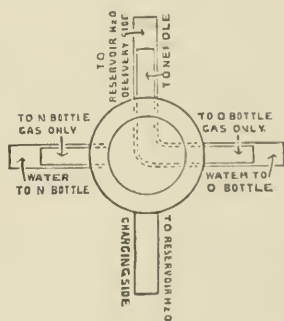


FIG. 2.

Diagram of the construction of Dr. Lister's gas and water regulator.

I have retained the very simple form of manometer used by Dr. de Carle Woodcock, which consists simply of a tall beaker containing a much narrower tube. The latter terminates above in a T-piece, one arm being connected to the gas delivery tube coming from the indicator,

the other to the gas delivery tube going to the needle. In the apparatus here exhibited, this manometer can be instantly disconnected from everything but the needle in the chest by the regulating tap, which closes all the water and gas channels by turning the pointer to any of the four places marked "Closed" on the dial.

This manometer has the advantage of allowing a free escape of gas should the pressure rise above the limit of the scale—viz., 15 c.m.

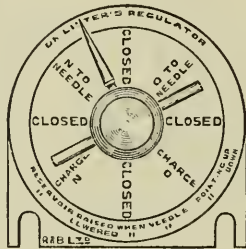


FIG. 3.

Front view of the dial of Dr. Lister's regulator.

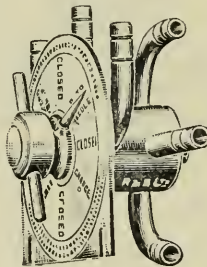


FIG. 4.

General view of Dr. Lister's regulator.

USING THE APPARATUS.

In small country sanatoria, and in private practice where gases are only seldom required, the travelling gas cylinder is often a source of annoyance. It is not uncommon when a gas cylinder has not been used for some time to find that there is no gas in it under pressure. This accident arises from various obvious causes, the chief of which is usually an insufficient closure of the valve. As in this operation only small quantities of gas are required, it is convenient to adopt Dr. de Carle Woodcock's plan of preparing the gas *in situ*. In charging the nitrogen bottle (which is usually the only one required during a refilling), the pointer of the controlling tap is turned vertically down-

wards to the word "Closed." When the indicator is pointing downwards for refilling the gas bottles, the reservoir bottle should also be brought down to the bottom of the standard. It is presumed that before filling a bottle with gas it was previously filled with liquid from the reservoir bottle on the last occasion when the apparatus was in use. The small bottle at the top of the standard carrying the gas bottles contains a solution of pyrogallic acid in caustic potash. This solution rapidly absorbs oxygen and carbonic acid gas from the air, and, when air is aspirated through it, delivers almost pure nitrogen, whose rate of absorption in the chest will be practically identical with that of absolutely pure nitrogen obtained in the ordinary gas cylinders. The solution is very corrosive, rapidly destroying any fabric and eating deeply into any wood upon which it is dropped. Care must be taken, therefore, to avoid reverse action, causing the solution to be blown backwards out of what should only be an inlet tube. This accident can happen if the reservoir bottle is raised and liquid allowed to run into the nitrogen bottle before cutting off the connexion to the aspirator bottle containing this solution. In recent forms of the aspirator bottle I have had a safety tap fitted in the neck. The aspirating bottle consists of a bottle of about 100 c.c., fitted with a stopper about 1 in. in diameter. This stopper is hollow and is drawn down at its lower end to a tube terminating in a bulb pierced with small holes. In one side of the neck of the bottle is fitted a glass inlet tube for the admission of air. On the other side is fitted an outlet tube for the discharge of nitrogen—that is, of air deprived of its oxygen and carbonic acid. In the side of the stopper a hole is made leading to the interior at such a level as to be opposite the air inlet in the neck of the bottle. On the other side a groove is cut on the outside of the stopper so that the interior of the bottle communicates with the outlet tube. The solution is poured into the bottle so as to cover the bulb at the end of the tube drawn down from the stopper. On producing a negative pressure by allowing the flow of liquid from the nitrogen gas bottle to the reservoir bottle, air enters the aspirating bottle by the inlet tube, passes through the stopper down to the bulb and escapes by the small holes in the side of the bulb. Here it loses its oxygen and carbonic acid, bubbles up through the liquid, and escapes up the groove in the side of the stopper, through the exit tube to the nitrogen gas bottle. Should the whole gas bottle of 600 oz. be emptied and the pressure attained in the pleural cavity be considered to be insufficient, the gas bottle can be refilled in a few moments without disturbing the needle in the chest.

When the pointer is now turned to the mark "Filling with N" on the dial the solution in the nitrogen gas bottle runs into the reservoir bottle and the nitrogen bottle is filled. If this is taking place while the needle is in the chest, the needle is only in communication with the manometer and during the refilling of the gas bottle no alteration of pressure occurs, and no further control of any part of the apparatus is required, so that the operator can watch the rhythmical movements of the pressure in the pleural cavity from time to time without disturbing the filling of the gas and vice versa. We will assume that the nitrogen bottle is now filled. To fill the oxygen bottle we will again assume that it was filled with liquid at the last operation performed. The pointer has to be turned downwards to "Closed," the reservoir bottle still in its low position, and the three-necked Wulffe's bottle is now charged for the manufacture of oxygen. Through the opening which is closed only by a cork is introduced a charge of about a drachm of a catalytic agent (peroxide of manganese). On the centre neck of the Wulffe's bottle is placed a two-way tap, one arm of which communicates with the atmosphere, the other with the oxygen bottle. I have recently added a gas-bag to the latter by a Y-piece between it and the oxygen bottle. A solution of an ounce of perborate of soda is now prepared in a beaker with water at about 70° or 75° F. The rapidity with which the oxygen will come off will depend on the temperature of the solution. If too hot the rapid effervescence is liable to be troublesome. The third neck of the Wulffe's bottle is fitted with a thistle funnel. This thistle funnel is bent so as to make the solution flow over the peroxide of manganese. On pouring the solution down the thistle funnel an effervescence occurs due to the setting free of oxygen. This idea is an application of the well-known oxygen baths made by Messrs. Zimmermann. The oxygen escapes to the atmosphere and washes out any air in the upper part of the Wulffe's bottle. On turning the tap in the centre tube to communicate with the oxygen bottle the liquid in the thistle funnel rises. The gas begins to collect in the gas-bag. When the effervescence has quieted down, and a good supply of gas has collected in the bag, the oxygen bottle may be filled. The pointer on the dial is now turned to "Charging with O," and liquid flows to the reservoir bottle, and the gas to the oxygen bottle from the bag. The oxygen bottle holds about 300 c.c. As it is not usually required except for the first operation, and then only until a pneumothorax is established, this amount is usually sufficient. As soon as the oxygen bottle is filled the two-way tap in the middle neck of the Wulffe's bottle is turned to

communicate with the atmosphere, and the pointer on the dial is turned to "Closed."

The operation of filling both these gas bottles, especially the oxygen bottle, is best done over a porcelain tray or other convenient place before taking the apparatus to the bedside or operating table. The charging of the apparatus is, of course, only a preliminary and is not part of the operation proper. For those who have always the facilities of gas cylinders at hand the attachments for the manufacturing of the gas are not necessary, and the apparatus can be charged in the ordinary way, but personally I have found them valuable in the out-patient room, in private practice, and in remote country sanatoria. A stock of the powders for preparing oxygen and of the solution for preparing nitrogen is easily kept. It is not necessary to change the pyrogallate solution frequently for preparing nitrogen. Dr. de Carle Woodcock, who first used this method of preparing nitrogen for this operation, told me, at the Armley Sanatorium, where I saw him use it, that he does not change it for several weeks. When exhausted the solution becomes very thick and black. The oxygen, which is much less frequently used, of course requires fresh powders and solution every time, unless sufficient oxygen remains in the gas-bag.

Both gas bottles being filled with their respective gases, the pointer is turned upwards to "Closed," and the reservoir bottle raised a little above the level of the gas bottles. The sources of supply of gas are now cut off from the gas bottles.

THE OPERATION.

I am accustomed to warm the charged apparatus before the fire and heat the room to 70° F. or over. If gas cylinders are used this precaution is additionally necessary, as the gas discharged therefrom has a very low temperature. I need not go into the *selection of the site* of operation; I would only remark that I consider delicate percussion by the finger a much more sensitive guide than the X-ray photograph for the selection of a spot free from adhesions. In percussing lightly one can usually detect shades of dullness which can be accurately diagnosed as adhesions due to slight thickening of the pleura, especially when confirmation is sought by the stethoscope. In my experience the anterior axillary line in the sixth or seventh space, or a spot a little behind it, is usually convenient. As a local anæsthetic I use ampoules containing 20 minims of a 2 per cent. solution of novocaine and $\frac{1}{10000}$ part of adrenalin. The ampoule is emptied into a sterilised hypodermic syringe. The skin

having been washed and painted with iodine, a point in the intercostal space a little behind the proposed puncture is selected, and half the contents of the ampoule injected along the course of the intercostal nerve. The needle is withdrawn and the remaining half injected vertically into the pleura at the site of the proposed puncture. I should not usually give heroin or other opiate previously to the operation, but I think very nervous patients with considerable cough may sometimes require it. I shall deal only with the puncture method. The question of the possibility of lung puncture on the one hand and the occasional blocking of a fine needle by pieces of superficial fascia on the other hand, led me to devise what I call a "half-blunt" needle introduced through the skin by means of a trocar and a cannula which is guarded so as not to be long enough to reach the pleura. This needle is made with a considerably blunter angle than the usual exploring needle. The end of it is closed and a lateral aperture is made as close to the end as

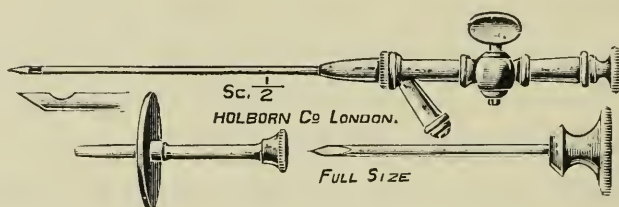


FIG. 5.

Dr. Lister's needle for artificial pneumothorax.

is mechanically possible—i.e., barely 2 mm. The little silver cannula has a plate fixed on at $\frac{1}{4}$ in. from the end and is introduced by a small trocar at a selected spot until the plate rests on the skin. The trocar is then withdrawn, leaving the little cannula buried $\frac{1}{4}$ in. in the superficial fascia, and, perhaps, in a very thin subject, reaching the intercostal muscle. The needle is then introduced through the cannula and pressed forward until it passes into the pleura, which is the toughest material likely to be encountered, and usually, as one knows from post-mortem experience, tougher than adhesions likely to be found in an area of soft lung. The needle enters the pleura with a distinct sense of something giving way. I would like to hear opinions as to the use of sharp or blunt needles (fig. 5).

In using the apparatus exhibited I stated that after filling the gas bottles the pointer on the dial was turned to "Closed." The needle is then only in communication with the manometer. When the needle enters the pleura the spongy lung is pushed forward by the blunt point,

and the lateral aperture almost immediately comes into communication with the potential pleural space. The negative pressure here existing sucks gas from the manometer. The fluid rises in the inner tube and begins to oscillate with the respiratory movement.

FILLING THE PLEURA.

The pointer on the dial being turned upwards to "Closed," and the reservoir bottle raised so that its fluid is an inch or two above that of the bottom of the gas bottles, and a negative pressure with rhythmical rise and fall having been observed, it is safe to introduce gas, but not before. In a first operation, in accordance with Rist's observations, I begin with oxygen. The pointer on the dial is turned to the mark "O to needle." The positive pressure, due to the level of fluid in the reservoir bottle, being above that in the oxygen bottle, at once causes gas under pressure to pass over the manometer, the liquid falls in the inner tube, and oxygen passes into the chest at the pressure marked on the scale. When a considerable negative pressure is found in the pleura, gas may be admitted at a less negative pressure instead of a positive pressure. In this form of manometer the scale consists simply of a strip of paper marked in inches or centimetres, or both, with a zero mark at the middle, and stuck on the outside of the large tube. The rate of flow is shown by the rise of the liquid in the graduated oxygen bottle.

Occasionally I have found that the gas runs very slowly from the oxygen bottle, and that, on raising the reservoir bottle slightly to increase the pressure, the greater positive pressure thus created in the manometer tube falls also very slowly. In such cases I think it is probable that there is some stickiness or some very soft adhesion in the neighbourhood of the commencing pneumothorax. By means of this regulating device one can easily secure the gradual "feeding" of oxygen into the cavity. I raise the reservoir bottle until the level in the liquid is 6 in. above that in the oxygen bottle. The fluid in the manometer tube runs down nearly to the bottom and I turn the pointer to "Closed." In such cases one observes a gradual rise of the fluid in the manometer tube. This is, I think, due partly to the gradual separation of the pleura by the gas under pressure. As soon as the pressure in the manometer has fallen a positive pressure is again created in the manometer by turning the pointer to "O to needle," then to "Closed" again, and so on. Gas is gradually fed into the cavity in this way, without

maintaining the stream of gas, in very small quantities simply by intermittent rises of pressure. In the last case which I did this condition existed, and I was able to introduce the whole bottle of oxygen with this simple means of control. When 200 c.c. or more of oxygen are introduced into the pleura some nitrogen may also be introduced. At a first operation the total amount of gas may be 300 c.c., but the patient's sensations and appearance must of course be a guide to the cessation or continuance of the first operation. One point which I suggest to the Section for discussion is as to the utility or desirability of introducing any oxygen at the first filling. The rapid absorption of oxygen necessitates early second filling. A proportion of nitrogen introduced at the end of the operation prevents the total disappearance of the cavity by rapid absorption. By the careful observation of the manometer it is often claimed that no risk of gas embolism from nitrogen exists even at a first filling.

Subsequent fillings are usually performed with nitrogen only, and the method is identical. The pointer is, in this case, turned to "N to needle," when the liquid runs into the nitrogen bottle and nitrogen is directed over the manometer tube to the needle.

A record should be kept of the pressure in the chest at the beginning and end of every operation for the introduction of gas. The amount of gas introduced should be measured and also recorded.

REFILLING.

When oxygen is used at the first operation, refilling should take place within thirty-six hours. The amount introduced on the second occasion will depend on the condition of the patient observed during the operation. When the patient complains of any feeling of tightness or pressure in the chest, or becomes at all cyanosed, or develops irregularity of the pulse, the admission of gas should be discontinued. Any severe symptoms from positive pressure can be immediately relieved by opening the tap in the needle. The pressure at which gas is admitted, apart from the question of breaking down soft adhesions, to which I have already referred, should be low enough to necessitate a proper time being spent over the flow of gas. To introduce 600 c.c. of nitrogen into a chest in which many adhesions are not present requires about twelve minutes, or even longer. If more gas is required, another 600 c.c. of nitrogen can be prepared in four or five minutes, and the whole 1,200 c.c. of gas introduced into the chest in twenty-five minutes

to half-an-hour. I have never introduced more than 1,200 c.c., but see no reason why larger amounts should not be introduced to secure a good compression, if no symptoms are produced. In regard to the pressure to be attained, it must be remembered that, on the other side, a presumably healthy lung or perhaps a less unhealthy lung has a negative pressure in its pleura. High pressures on the diseased side may cause cardiac displacement, and considerably disturb the vascular conditions existing on the healthy side. The lung on the side on which the operation is being performed had originally a negative pressure which would depend on the amount of retraction possible. In one case of marked general infiltration I found an almost complete absence of adhesions, and the negative pressure before commencing the introduction of gas was minus 21 cm. In other cases there may only be a negative pressure of 3 cm. to 5 cm. At any moment during the operation the pressure attained in the chest can be observed by means of this regulating device by turning it to the word "Closed," whereby the needle is placed only in communication with the manometer. If adhesions are being separated by using an intermittently increased positive pressure in the gas tubes the pressure in the manometer will be seen gradually to fall while the indicator is at the word "Closed," until it becomes stationary, except for the rhythmical rise and fall during respiration.

I believe that in ordinary cases of strongly marked unilateral infiltration without adhesions, a good compression can be obtained by positive pressures not exceeding 10 to 15 cm. Dr. Morland is of opinion that gas at positive pressure is absorbed very rapidly. It must be remembered that the new pneumatic pressure on the selected side of the chest has to be measured by adding together the original negative pressure which has been annihilated and the new positive pressure which has been created. I suggest as a point for discussion that the utility or risks of high positive pressures should be considered. As a basis for the discussion of this point our object should be kept in view. It is to compress and immobilise the lung which is most diseased. In my own experience the amount of gas introduced and the pressure attained without severe discomfort to the patient can only be judged at each refilling. My rule would be to introduce as much as possible and to attain a positive pressure up to the commencement of sensations of distension and discomfort. This is a matter of observation and it should not be necessary to open the relief tap on the needle to allow the escape of gas and the reduction of pressure. The pressure attained and the patient's sensations are more important observations than the

amount of gas introduced. But the amount of gas introduced to attain a certain pressure in the chest is to some extent an indication in a well-established case as to the amount of gas that will be necessary to attain the same pressure after the same interval, at a subsequent refilling in a patient who runs a normal course. During the interval absorption takes place and the pressure falls. The gradual absorption of nitrogen seems to vary, however, in the same patient after each filling. This is true even when the pneumothorax has been established as long as twelve months. X-ray examinations are of value throughout the course, and should be made at each refilling if possible.

INTERVALS BETWEEN REFILLINGS.

No general rule can be laid down as to the intervals which should elapse between each two refillings, except this, that the intervals

ARTIFICIAL PNEUMOTHORAX.

Date	Gas	Quantity in cubic millimetres	Initial pressure	Final pressure
1913				
March 26	{ Oxygen Nitrogen	175	Centimetres - 2.5	Centimetres + 4.0
April 2		525	- 3.5	+ 3.0
" 9	"	580	± 0.0	+ 4.0
" 18	"	650	+ 0.5	+ 4.5
May 2	"	850	- 0.5	+ 4.5
" 21	"	850	- 1.5	+ 4.5
June 9	"	900	- 1.5	+ 4.5
" 26	"	900	- 0.5	+ 4.0
July 8	"	500	± 0.0	+ 4.0
" 31	"	900	- 2.5	+ 2.0
August 19	"	1,000	- 2.5	+ 2.0
September 9	"	1,000	- 2.0	+ 2.5
October 9	"	1,000	- 1.0	+ 3.0
November 7	"	1,050	- 3.5	+ 0.5
December 13	"	1,000	- 3.5	- 0.5
1914				
January 22	"	1,150	- 6.0	- 0.5
March 2	"	800	- 0.5	+ 0.5
April 29	"	1,000	- 2.5	+ 0.5
June 5	"	1,050	- 2.5	+ 5.0

had better be too short than too long. My opinion is that where the disease on the side selected is of a chronic nature with some fibrosis, more frequently repeated fillings and a more sustained pressure are desirable, especially in the early stages. In such cases pleural pockets are frequent and adhesions commonly present. The

treatment of cases of this kind is a matter also for our mutual consideration. Small quantities of gas will cause a considerable pressure when the artificial pneumothorax is made in such a pocket. By more rapid repetition and keeping the pressure well sustained the tendency is towards the gradual separation of more pleura. Although the rapid separation of adhesions by pressure, with the production of a sharp pain, has been described, I think the method of keeping up a sustained pressure perhaps of 7 to 12 cm. positive pressure is preferable, although there is no doubt that in some cases the sudden separation of the pleura is not followed by any bad results. Adhesions are the most frequent cause of failure. The excuse for the open operation is the presence of adhesions. In each case judgment must be exercised as to the interval to be allowed. In a case of progressive infiltration, where the first manometric observation shows a marked negative pressure and adhesions are absent, rapid compression of the lung is possible by the introduction of considerable quantities of gas by the end of a week. In two or three sittings a litre of gas may be reached as the amount to be introduced, of course subject to the patient's tolerance. I may illustrate the history of a case successfully treated by the actual table of fillings of a patient under the care of Dr. Morland and myself.

DURATION OF TREATMENT.

The minimum duration is a question upon which discussion is certainly possible. My own opinion would be that an average duration is at least two years in a successful case. Subsequently such cases should be seen at least three or four times a year for some years. The absorption of nitrogen is slower and the intervals grow longer as compression becomes well established. Manometric observations at the beginning of each operation show whether the pressure is fairly well maintained. As a rule the rate of absorption of gas diminishes, but this, however, is certainly affected by the amount of exertion which the patient undertakes as well as by the pressure attained. In the table which I have just brought before you, the patient was addicted to mountaineering and ski-ing on slight provocation. In one letter, Dr. Morland stated that a few months after the commencement of treatment he climbed 3,000 ft. above Arosa without distress. The healthy portion of the lung may expand even after collapse continued for several years. What risk is run on the one hand by too early a discontinuance and on the other hand by too long a compression? What are the indications for cessation of treatment?

SELECTION OF CASES.

My own feeling is that a great many cases are being treated by this method in which, in spite of a temporary reduction in toxæmia, no permanent benefit can be expected. As an indication for the treatment, I would suggest that the typical case in which the operation is demanded is that of progressive infiltrating disease which is markedly unilateral, and in which little or no tendency to fibrosis can be observed when the patient has been placed under good hygienic conditions for two or three months, with all the precautions which obtain under the recognised methods of sanatorium treatment. Another class where I have found this operation completely successful is the markedly hæmorrhagic, especially where the hæmorrhages occur frequently in a soft, spongy lung. Softness of the lung is, I think, the most important factor in successful treatment by this method. As I have said elsewhere,¹ this is an operation for cases with feeble resistance and not one for cases with chronic fibrosis. The use of the X-rays in conjunction with clinical examination is a help in estimating the softness of the lung. Cavitation is not a bar, provided that the lung is in a soft condition below the area of the cavity and that excessive adhesions are absent. In other words, where active fibrosis is going on, and therefore strong adhesions are probably present, we have no reason to believe that compressing a few patches of the remaining healthy lung is going to produce much effect. Gas pressure has little effect on a tough old chronic fibroid phthisis. But, on the other hand, remarkable cases of recovery have been recorded from extensive disease with acute symptoms. The contra-indications are naturally the reverse. Extensive bilateral disease with cavitation, chronic fibroid disease, and the usual constitutional indications against any operative shock, are the chief.

I see no reason why a bilateral operation should not be done with discretion, but have not yet selected a case.

There is one point in the selection of cases which is perhaps to be noted. The patient's residence and the possibility of his being able to obtain refills after leaving the institution in a convalescent state, have to be taken into account. In some great centres of population no facility as yet exists for the patient to obtain the after-treatment which is necessary. It is difficult to persuade a patient who has greatly improved as a result of compressing his lung, that it is necessary for

¹ *Clin. Journ.*, October 21, 1914, p. 625.

him to take long journeys to have another injection when he feels quite well. More patients suffer from not being refilled sufficiently often and for a long enough period, than from having the operation or too frequent refillings. Professor Saugman has been quoted as saying that his exceptional experience teaches him that the rule should be, when in doubt, "go on." But ultimate failure will result from the patient's non-attendance to be refilled at the proper time for any reason more frequently than from any other cause.

COMPLICATIONS DUE TO THE OPERATION.

I have not seen any of the major accidents which have occurred in those who have had a large experience. Gas embolism is a grave danger, which is to be avoided by the intelligent observation of the manometer, and, according to Rist, by the use of oxygen, especially at the first operation. I think, also, that the use of such a form of needle as that I have brought before the Section to some extent also obviates this risk. But the safety of the patient really depends upon the continued observation during the whole time of the operation of the rhythmical rise and fall of the fluid in the manometer with the movements of respiration. The so-called pleural reflex is an accident which may occur in this form of puncture as well as in the ordinary tapping for effusion. I think the anæsthetising of the pleura is a means of avoiding this risk generally. One condition I have seen in a case at present under my care is the conversion of an artificial pneumothorax into a natural pneumothorax by the giving way of a weak spot in the lung. The operation was successful for five months, and two weeks after the last administration of gas an attack of dyspnœa occurred and there is now a communication between the pneumothorax and the air passages, and the lung has not re-expanded. The patient has relapsed seriously. At the administration before this occurred 800 c.c. of nitrogen had been introduced, and the final pressure was 14 cm. I should be glad to know if anyone has met with this accident. I am in doubt as to whether it could be ascribed to an excessive pressure on the outside of a weak spot in the lung or merely to a destructive lesion due to disease.

Pleurisy and pleural effusion occasionally occur, but I have only seen a transient pleurisy once.

RESULTS OF THE TREATMENT.

Perhaps the most careful table is that so frequently quoted, which was given by Saugman in his paper read before the last International Medical Congress in London.¹ As his experience is quite exceptional and most carefully reported, I once more quote his figures. In treating 141 patients at least nineteen months before speaking, he had been successful in producing a pneumothorax in ninety-five instances. In forty-three cases he considered that he had failed or that the pneumothorax was too small to be effective. He considered that 100 of his cases were suitable selected cases; the remainder were not cases that one would have selected for treatment, but cases in which life was threatened by serious progressive disease or complications of a tuberculous nature. These he classifies as having been quite hopeless cases in which the treatment was adopted to give them a chance. Of those cases which would be considered as suitable for pneumothorax treatment, 100 in all, sixty-four cases were successful as regards the induction of the artificial pneumothorax, and in thirty-six the pneumothorax failed. The after-history of the cases where the pneumothorax was successful as compared with those where the pneumothorax failed affords a very remarkable piece of evidence. All these patients were under the same sanatorium conditions, and they were patients of practically the same class, where the lung on one side was much less affected than the lung on the other side. I quote his table:—

(1) FIT FOR PNEUMOTHORAX TREATMENT.

	Pneumothorax effective (64)	Pneumothorax failed (36)
Able to do ordinary or lighter work ...	32	8
Unable to work on account of tuberculosis ...	18	12
Dead from tuberculosis ...	12	14
Unknown ...	0	1
Dead from acute complications ...	2	1

(2) NOT CONSIDERING THE LAST THREE, THE PERCENTAGES ARE AS FOLLOWS:—

Able to work ...	50.0	22.2
Unable to work ...	28.1	33.3
Dead from tuberculosis ...	18.7	38.3
Freed from tubercle bacilli ...	50.0	8.6

Some of those present to-day will be able to add points from their own experience, both in regard to the successful and unsuccessful cases. I am inclined to think that Saugman's figures are a reliable guide as to the value of the method. Only a small proportion of cases are really

¹ *Internat. Congr. Med.* (XVII), Lond., 1913, Sect. VI, Medicine, pt. ii, p. 463.

suitable, and I should hesitate to say that every case of unilateral disease should be treated by this operation. Easy and simple as the operation may be, the changes produced in the intrathoracic conditions are considerable. Although the charm of doing something active for consumptive patients must appeal very strongly to all of us, yet misguided enthusiasm in this, as in so many other treatments for tuberculosis, results only in an excessive loss of confidence. The greater question involved in every case of open tuberculosis is easily lost sight of. The immediate subsidence of symptoms is no doubt a great gain, but in every case we must remember that there has been a complete breakdown of the immunising machinery. We know yet of no means of restoring that machinery except by careful and prolonged hygienic treatment. Clinical impatience is as bad for the victim as it is for our professional self-respect. I see no object in treating large numbers of cases for the sake of counting our punctures.

CONCLUSION.

I feel I have been very brief in dealing with the general aspects of the question. No doubt they will be dealt with by those taking part in the discussion. The apparatus I have exhibited claims only to be self-contained and simply controlled. Being self-contained, it may appear complicated to those doing a large number of these operations and keeping a constant reserve of compressed gas at their disposal. But it has been found useful in several country institutions where only carefully prepared cases are treated by this method. It is also convenient for occasional private operations. It must be remembered in comparing it with other apparatus, that the latter require gas cylinders, and their controls and connexions, which are considerable additions. I also think that regulation from one point during the operation is of advantage in enabling the operator to keep his attention fixed on the manometer. I must apologise for having gone over many details in describing the apparatus and the operation, which are matters of every-day familiarity to many of those present. But apparatus, after all, is a trifle. The manner of its use and the methods of its users are all that matter. I would mention again the points upon which I would like to hear opinions expressed, without any desire to limit discussion:—

(1) The use of oxygen.

(2) The selection of sharp, blunt, or guarded needles. I suggest

that my own needle is a compromise between these methods and is very generally applicable.

(3) The uses of high pressures. I would define a high pressure as anything over 5 cm. of positive pressure.

(4) The treatment of cases presenting strong evidence of adhesions and pleural pockets.

(5) The indications for cessation of treatment by compression.

(6) The causes of failure, immediate and remote, including the selection of cases.

DR. JANE WALKER: I suppose we should all be agreed that the ideal case in which to produce an artificial pneumothorax, or, in other words, to put the bad lung completely out of gear as far as working purposes are concerned, would be one in which the disease was absolutely confined to the one lung. But we all know how very rare such cases are, some of us even going so far as to regard a purely one-sided case as practically non-existent. Still the fact remains that, in weighing the pros and cons, the condition of the other lung has to be taken into consideration. Is the disease in the good lung in an active state? Is it progressing? Is it so extensive as to lead one to suppose that the extra burden thrown upon it by doing double work for a time is likely to strain it to breaking point? Having settled the local condition to our satisfaction, there is the general state of the patient to be taken into consideration. Should the procedure be carried out when there is serious laryngeal mischief, or a renal or abdominal complication? What prospect of success is likely to be attained in the presence of a severe hæmoptysis? Then the state of the nervous system has to be considered. Is the patient too timid, too lacking in self-control, too apprehensive and "jumpy," for the operation to be undertaken without fear of injury? Finally, the circumstances of the patient must be taken into careful consideration. Has he the means to continue the treatment till the end—a period of time that will probably extend to two years, and may last much longer, and which, moreover, may be marked by complications which will require skilled treatment? If the patient has means, is he under the care of someone who, while quite recognising the marked improvement produced by the treatment, either refuses himself to go on with it or recommends that there should be no further refills? If he has not the means, is he helped or hindered by the Insurance Committee or other local body responsible for his treatment? Is he under the care of a tuberculosis officer who knows

what he is doing when he expresses an opinion as to artificial pneumothorax and the continuance of the treatment, or is the patient the unfortunate victim of a man whose opinion is expressed with that freedom which a person totally unhampered by a knowledge of the facts is so fully competent to do, and who refuses to allow him to undergo any further treatment?

From March, 1912, to January, 1915, I have had fifty-six cases under my charge, and they will, I think, fairly exemplify the various considerations to which I have just referred. Of these cases twenty-nine were women and twenty-seven men. They varied considerably in their ages—the women from 14 to 55, the men from 18 to 42. All were aged under 40 except one woman and two men. Of the twenty-seven men ten have died, and of the twenty-nine women, seven. The mortality has therefore been, both sexes included, 30 to 35 per cent. : men alone, 37 per cent. ; women alone, 24 per cent. There has been no fatality, nor even any untoward effect, as an immediate result of either the primary initiation of the artificial pneumothorax or any subsequent refill. Twenty-three cases are still under treatment, twelve of them being still in residence at the sanatorium. Of the other eleven, nine attend either at the sanatorium or at my house in London for their refills, and the other two are having them at the Delamere Forest Sanatorium. Of these eleven cases, six are leading an ordinary life and carrying on their usual avocations of blacksmith, gardener, instrument maker, post-office clerk, and houseworker respectively ; the sixth, a salesman, is perfectly well, but he was sent to me from Canada, and he is returning there when he has finished his treatment. The remaining five are all up and about ; one of them will soon be fit to undertake work as a nurse again, another as a servant ; one will need further sanatorium treatment because she is showing some fresh signs in her hitherto good lung ; the remaining two are in poor health, but able to keep about and do a small amount of work. Of the twelve cases still in residence, in five the disease was acute and progressing rapidly, with high fever and loss of weight and strength. Both lungs were involved in four of these five cases, the remaining patient showing no signs of disease on the good side either with ordinary physical examination or by means of X-rays. The other seven cases were more chronic in character, with disease of longer standing, and in three the disease was limited to one lung.

Of the remaining thirty-three, two had severe laryngeal complication ; one patient—an old-standing case—who had been treated in

1906 for severe laryngeal tubercle, with small lung involvement, had a pneumothorax produced in April, 1912. Her lung was collapsed for about two years, during which time she lived at home and did a small amount of work. She died about two months ago. In the other case treatment was started in April, 1914, and she is still going on with her refills. Her last report to me was at the end of February, and was, so far, highly satisfactory. In four cases we have produced a pneumothorax on account of severe hæmoptysis. Two of these cases died and two recovered. An artificial pneumothorax was produced in the two patients who died while the hæmorrhage was going on—indeed, one case had a severe hæmoptysis during the first injection of the gas. In this case there was no further hæmorrhage after the first refill and the lung appeared to be fairly well collapsed, but the temperature rose to 104° F., the dyspnœa increased, and the patient died at the end of a week. In the case of the second patient who died, there was no more hæmorrhage (his hæmorrhages had been excessive, 1½ pints or so at a time), but his temperature, too, rose to 104° F., and remained mostly between 104° F. and 102° F. till death on the eleventh day. In both these cases, seeing that the disease was progressing, we ought not to have waited, but to have produced a pneumothorax sooner. But these cases occurred over two years ago, and were the tenth and sixteenth, respectively, that we had treated by this method. In the case of one of the other two, we waited till the hæmoptysis ceased and the temperature came down to normal before producing the artificial pneumothorax. In the last case the patient had been at the sanatorium for some months, and been improving greatly when, in the early part of the summer of 1912, she had some repeated hæmorrhages and showed signs of fresh disease in the other lung. She did exceedingly well and left in June, 1913, for domestic service. Her chest wall fell in and shoulder dropped, the bony framework of the chest adapting itself to the collapsed lung.

There appears to be some difference of opinion amongst practitioners as to whether the procedure should be undertaken when *active* mischief is in progress in the bad lung. I gather this from one of my communications with a chief tuberculosis officer who refused to sanction the necessary expenditure for a patient to come up to have his refills. "I hold," says he, "very strict views with regard to the type of case which should be selected for artificial pneumothorax, and in my opinion the case in which there is active disease in either lung is not suitable for this method of treatment." He further adds: "The aim of artificial pneumothorax is to secure lung rest, and I do not think that this can

be secured unless the treatment is carried out in active cases in an institution and the patient is kept rigidly at rest." Certainly, several of the very best cases we have had have been those in which the disease was acute and active, and in certainly one case, if we had waited till the disease was quiescent, we should have lost the patient.

With regard to disease in the good lung, the safest procedure is to very gradually collapse the bad lung by small, frequently repeated refills, and it has been my experience that, when this is done, the disease in the opposite lung gradually clears up, *pari passu*, with the marked improvement in general health.

It has been said, by someone well qualified to make the statement, that artificial pneumothorax is the greatest advance in the treatment of pulmonary tuberculosis made in the last ten years, a statement with which I heartily concur. For, alas, I can think of many patients prior to 1912 who might have been alive now, and living useful lives, if we had then had this method of treatment at our disposal.

Dr. DE CARLE WOODCOCK: While disclaiming any wish to be too biographical, I may give details of one case of marked bilateral disease as an example of many others. The daily sputum of the patient has been reduced from 13 oz. to 1 oz., and the symptoms, as in Dr. Jane Walker's cases, have declined in almost equal ratio. The patient has relapsed whenever treatment has been discontinued, but probably he has gained several years of life. No cure can be promised in such cases; but, apart from slight surgical conditions, such as appendicitis, can any disease be said to be cured? I have operated upon many patients, in the early as well as in the late stages of the disease, and I recognise that the dangers and the treatment differ. It is possible to be quite successful with one set of patients and sick at heart concerning others.

The dangers of the operation may be considered as (1) immediate and (2) remote. The remote dangers, including sepsis and strain on the lung left to do the work, I must leave on one side for the present and confine my remarks to the immediate dangers. These are: (i) shock—i.e., irritation reflex; (ii) gas embolism; (iii) clot embolism; (iv) asphyxiation from direct pressure of gas in the intrapleural space.

Shock.—Brauer and Spengler record and lament the deaths of patients of fine physique and limited disease. These deaths are very significant. The comparatively healthy nerves of the pleura, as they are found in early disease, are very sensitive to injury, and shock

easily results. The exploring syringe often brings out this tendency to shock.

Embolism.—Brauer and Spengler, in criticising post-mortem reports of deaths presumably due to gas embolism, recognise the difficulty of diagnosis before death, and refuse in many cases to recognise the findings after death, many vague symptoms and signs not referable to anything definite have been supposed to indicate the occurrence of gas embolism. Brauer and Spengler regret that a skilled pathologist was seldom present at the post-mortem examinations. The conclusions one reaches after reading their reports is that death might in most cases have been due to causes other than gas embolism. But however this may be, there have been but few deaths recorded. How many of us, I ask, have personally known of one case? I have in my own practice met with symptoms of gas embolism—so-called—where no gas has been injected. The truth is that we have been reared in fear. The fear is traditional and has lived upon ancient accounts of obsolete operations. Sir Berkeley Moynihan tells me that there has been no case (in his knowledge) of gas embolism among the wounded for whom gas injections into the tissues have been employed. Professor Jamieson tells me that there should, *a priori*, be little danger of embolism in the tissues outside the lung, and that the anastomosis between the systemic and the pulmonary circulation is not likely to be a source of danger. Any gas in anastomosing channels to do harm would have, as it were to swim against the stream. The danger of gas embolism may reasonably be considered as but one among the heptarchy of dangers. It is slight in tissues external to the visceral pleura; it is almost negligible outside the costal pleura. The vessels large enough to be canalised by a Saugman's or a Lister's needle are deep in the lung. A lung may be so grossly diseased, its veins so stiff and thickened, that canalisation or tearing across might, it would seem, easily occur if the needle were pushed in deeply, especially if the puncture be near the root of the lung. In such cases, also, a blood-clot might be mobilised by any traumatic disturbance and sucked into the moving blood-stream. The suction, of course, comes from the powerful *left* ventricle.

Asphyxiation.—There is one danger which is not referred to in the literature of the subject. When both lungs are so grossly diseased that they can bear no interference, the sudden application of a large volume of gas to one lung will in effect put the patient in the position of a drowning man. Cyanosis amounting to blackness of visage, unconsciousness, convulsions, death, may follow.

I may also refer to the danger of alveolar gas embolism, to Saugman's fatal case of gas embolism due to alveolar air, and to the researches of Brauer, which tend to show that in primary puncture the lung is always scratched.

I consider that all the manometer signs of safety may prove deceptive; on the other hand, one may sometimes have to pass the signals, or apparently so. With regard to figures, three thousand operations have been done by myself, Dr. J. A. M. Clark and Dr. Gebbie, of Killingbeck, without one fatal accident. One rule all must observe—never to dip deep with the needle.

Dr. LISTER (in reply): I have been very interested in the striking temperature charts shown by Dr. Jane Walker. So far as the immediate effects of artificial pneumothorax go I am quite in accord. I can exhibit a chart which I have with me this afternoon of a man who was admitted under my care at the Mount Vernon Hospital in a moribund state from the Friedenheim Home. The chart shows exactly the same characters as that exhibited by Dr. Walker, and the man recovered in a remarkable manner up to a certain point. He became sufficiently well to travel to the Benenden Sanatorium, where he secured quiescence for the time being. But he ultimately relapsed and died with extension of the disease to the other lung. Artificial pneumothorax undoubtedly relieved the strain on the patient's failing resistance, but the problem of curing phthisis is not to be settled by this physical means. The underlying difficulty is the fact that in these patients a complete breakdown of the immunising machinery has occurred. Their special liability to the disease continues. The relief of the strain on this broken-down machinery is what is accomplished by the operation, and this gives time. It may enable a patient to recover, as we hope, and as Dr. Walker has shown. But the fact that the machinery has broken down in an individual leads us inevitably to fear that similar conditions of environment, collateral diseases, or other causes, may lead to a similar breakdown again. At the same time, this treatment should not be denied to cases of low resistance when they are placed under the best hygienic conditions.

In regard to what Dr. de Carle Woodcock has said, I confess myself not quite able to follow his argument. Although in Dr. de Carle Woodcock's hands there may be little risk in passing gas into the chest when the manometer is not swinging freely, I would nevertheless hesitate to do this. I like to be sure that I am in the pleura,

and that I have compressible lung beyond my needle. I think that my half-blunt needle prevents the danger of pricking the lung if used with the guarded trocar that I have described. In regard to the absence of gas embolism mentioned by Dr. de Carle Woodcock as having been observed in the military hospitals, I notice that he does not mention in his remarks what gases were concerned. [Dr. DE CARLE WOODCOCK: Hydrogen peroxide, oxygen, and air.] Hydrogen peroxide is oxygen and water when it comes in contact with the tissues, oxygen is oxygen, and I consider air only 20 per cent. less dangerous than nitrogen. I am convinced that nitrogen is much more dangerous than oxygen, from Rist's observations. Personally, I should use oxygen at a first operation, until at any rate I have established a clear cavity of some 200 c.c. or more.

I believe that this operation has a great future before it, and that its use may be defined as consisting in gaining time while improving the patient's resistance by the ordinary hygienic methods. The cases to be selected for this mode of treatment should be those of proved low resistance where the disease is markedly unilateral, and not cases of chronic fibrosis or disease at the earliest stage. I think the number of cases requiring the treatment will be found to be very limited, probably much less than 10 per cent of the average sanatorium cases.

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(Prepared by Mr. A. L. CLARKE, Assistant Editor)

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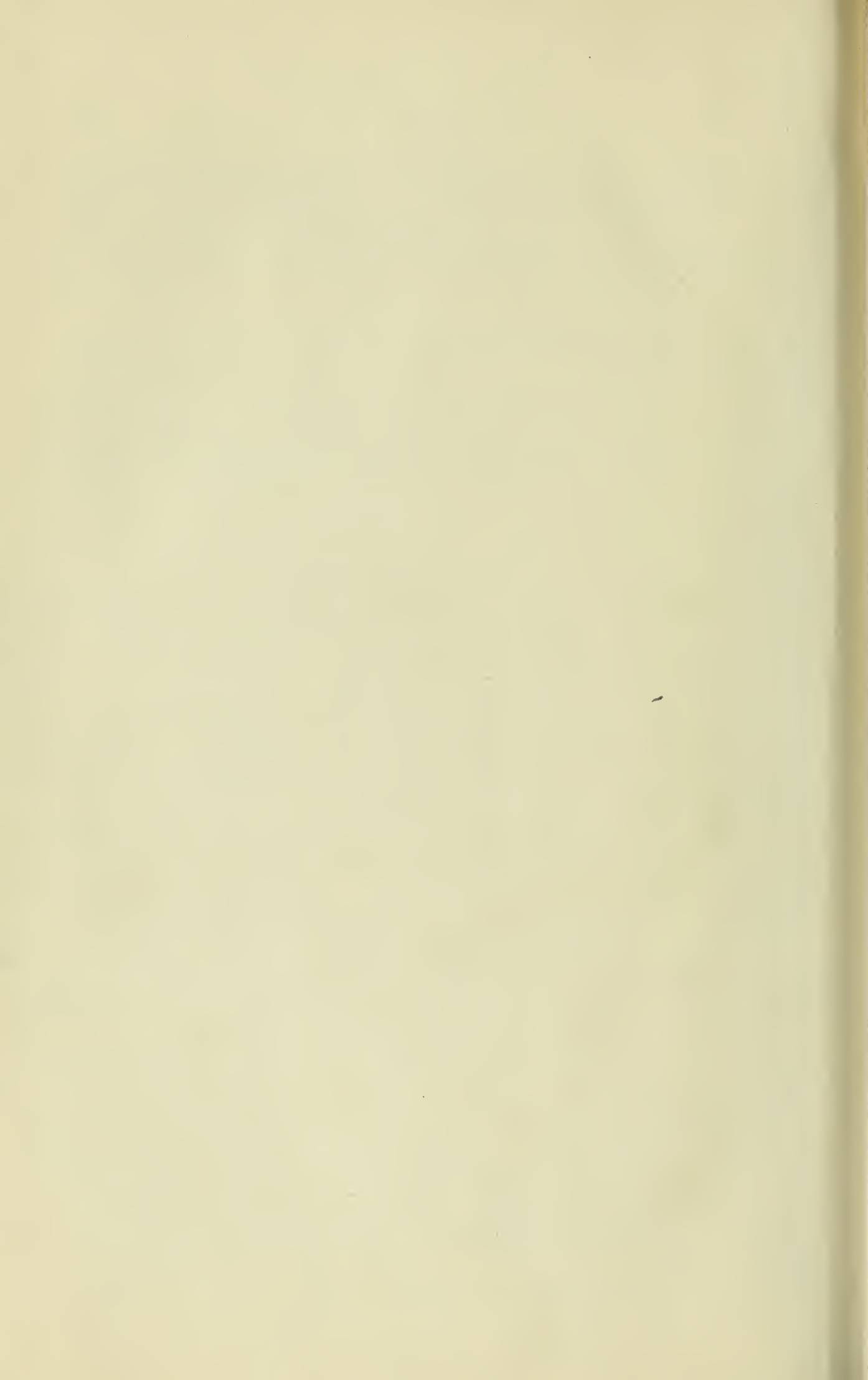
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